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The Profession and the Poultry Industry

NO section of agriculture has shown such a phenomenal expansion during the last few years as the poultry industry which has now become the second most important branch, producing some 18 per cent. of the total agricultural output of the country, and with a turnover in 1959 of £280,000,000.

That this expansion has been possible in the face of the tremendous disease hazards associated with flocks of vast size maintained under highly intensive forms of management is to the credit of the relatively few veterinary research workers, diagnosticians and field advisers. The profession as a whole, however, has taken less interest in poultry, so far, than could be wished.

A few of the larger poultry organisations are now employing their own veterinary advisers, but there are still a number not yet quite large enough to justify this step and they, together with the many farms on the group system, require help urgently.

It is doubtful whether existing diagnostic laboratories can continue to cope with the spate of material reaching them, and presumably the increase announced by the Ministry of Agriculture in *post-mortem* examination fees is an attempt to control the flow. Here is the profession's opportunity to help, and with the increase in flock size, "on the spot" diagnosis and advice is essential. There are inevitable delays in laboratory diagnosis and in many cases laboratories, if relieved of the mass of routine material, could better devote their energies to *ad hoc* investigations of obscure problems.

It has been argued in the past that poultry do not lend themselves to inclusion in general practice and reasons have been advanced which, although they may have had some relevance in the past, are no longer valid.

Up to the end of the last war some 80 per cent. of poultry produce came from flocks of under 100 bird size, mainly kept by the farmer's wife for pin money; but in the last 6 years, the number of specialised units has trebled and the poultry industry has felt the impact of "intensivism" to a greater extent than any other form of livestock farming. Breeding flocks with expensive and highly scientific genetical programmes, involving many thousands of birds, are common. Battery units for egg production holding 20,000 and in some instances 50,000 birds, are by no means rare, whilst in the broiler industry, although the average size of each supply flock is 15,000 birds per crop, the daily throughput of a processing plant serving a "group" may be 25,000 birds. There are a few plants with an annual capacity of nearly a million birds. In the highly organised business of chick production there have been developments culminating in "walk-in" incubators giving, in individual plants, an annual output of 12½ million chicks.

It has further been said that the low individual value of the fowl does not justify clinical examination or individual therapy. On the contrary, the low individual value of the bird is a great advantage and for this reason the majority of poultry farmers, realising that the health of the flock is of more value than the well-being of any of its component units, are always prepared to cull rigorously in the hope of checking the spread of disease. Furthermore, the advent of coccidiostats, antibiotics and similar drugs has made it possible to carry out mass therapy *via* the food or drinking water. There are many, however, who would wish to see this development of "pharmo-nutrition" discussed regularly with the profession so that our knowledge could be utilised fully by the industry and the poultry farmer.

The third and weakest argument has been that poultry diseases can be diagnosed only in the laboratory by specialists. This is true of certain bacterial and viral infections and, important as they may be, analyses of *post-mortem* results indicate that the majority of losses are associated with outbreaks of coccidiosis, avian leucosis, and other conditions which can be diagnosed with reasonable accuracy by simple field *post-mortem* examinations. The efficiency of most drugs is in direct relation to the speed with which they are applied after infection. There is a further advantage in that the lay-out of most poultry farms lends itself to field experimentation, with adequate control groups, and the work as a whole can be rewarding.

Some practitioners appear to feel themselves on unfamiliar ground when dealing with avian disease. Yet there is nothing mystical about poultry ailments and our basic training should make us equally competent to deal with avian diseases as with those of other species, provided that we are prepared to apply ourselves to the subject. It must be accepted that the

(*Concluded at the foot of col. 2, page 983*)

Congress Papers

SYMPOSIUM ON POULTRY DISEASE AND THE PRACTITIONER

(1) Infectious Diseases

BY

J. D. BLAXLAND

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SUMMARY.—This paper is a brief account of the more important infectious diseases causing mortality and unthriftiness among fowls, turkeys and ducks, with comments on the control and treatment of outbreaks in the light of recent research and experience.

The introductory paragraphs of the paper are an attempt to ensure that the economic importance of infectious diseases in the context of this symposium is not exaggerated unduly. Attention is drawn to the influence of genetical factors, nutrition, general husbandry and environment on the health and profitability of poultry keeping.

Veterinary surgeons in practice wishing to extend their activities into the poultry field are reminded of the difficulties of establishing a definite diagnosis in many outbreaks of non-specific mortality and unthriftiness among poultry and the impossibility of doing so accurately without some understanding of modern poultry management, nutrition and hygiene.

THE organisers of this programme did well to regard infectious diseases as only one of the factors causing economic loss in the poultry industry to-day. To have singled out diseases as the sole topic worthy of consideration by the veterinary surgeon would have been misleading, and might result in the false assessment of field problems and an incomplete diagnosis in certain cases. Ill-health and unthriftiness in poultry flocks due to non-specific factors are often of greater economic significance than is mortality caused by infectious disease. Some of the more common infectious diseases prevalent to-day will be discussed in this paper but their significance should not be exaggerated.

The relative importance of the parts played by a disease agent and the environment of the host is often a matter for speculation, but the training and experience of the veterinarian should enable him to assess correctly the significance of the various clues which may provide the key to the problem of an outbreak of disease in poultry. However, it is not always possible to define a specific bacterial, viral or parasitic agent in any one outbreak of disease and to apply the appropriate remedy for its treatment and control. Disease, under the intensive conditions of management by which the modern poultry industry operates, is not simply a matter of specific infectious agents and their treatment. The problem in most outbreaks is posed by the question, "What mistakes or errors have been committed on this particular plant to account for the inability of the flock to withstand the stresses and strains of living in this particular environment?"

Paradoxical situations are continually arising under field conditions: mortality and unthriftiness from caecal and intestinal coccidiosis in one flock of broiler chickens, necessitating continual expenditure on drug therapy, whereas on other plants there is no serious disease problem, despite the ubiquitous distribution of the causal parasites; or respiratory diseases which cause debility, depressed egg production, fertility, and hatchability on only a small number of farms despite their obvious presence to a slight degree among poultry of all ages in so many other flocks; or the serious spread of fowl typhoid during the last 10 to 15 years from areas in which it has been long recognised as an endemic disease to many other parts of the country where previously it was almost unknown and where different environmental conditions prevailed.

The achievements of research workers, combined with the skill of the manufacturing chemists have produced a steady succession of antibiotics and drugs which have enabled the poultry farmer to expand his flocks under increasingly intensive conditions without the disastrous loss from disease which might otherwise have occurred. But the almost universal use of drugs and antibiotics in proprietary rearing mashes, crumbs and pellets has also complicated the picture as it tends to suppress evidence of infection, distorting the characteristic manner in which diseases normally occur and the symptoms by which they might otherwise have been recognised, thus increasing the difficulty of diagnosis. Strains of *Salmonella typhimurium* resistant to antibiotics have already been produced under experimental conditions (Garside, Gordon & Tucker, 1960). Similar experiences have been reported by Williams Smith & Crabb (1956; 1957) working with calves, pigs and fowls infected with *Escherichia coli*. These observations are a warning that the successful use of drugs and antibiotics for the control and prevention of disease may bring further complications in their train in the future.

The veterinarian investigating outbreaks of disease among his clients' poultry must not overlook the influence of genetical factors, nutrition and environment of the flock.

Hutt (1958) mentions a number of conditions in poultry in which genetical resistance or susceptibility have been demonstrated. Williams Smith (1956) demonstrated considerable variation in the susceptibility of different breeds to fowl typhoid, heavy breeds proving more susceptible than light breeds. Selective breeding for resistance to disease has been explored seriously by only a small number of

* A symposium of papers presented to the Annual Congress of the British Veterinary Association in Glasgow on Tuesday, August 30th, 1960.

workers. The achievements of Hutt at Cornell, and those of the team of veterinarians and geneticists at East Lansing, working on leucosis and fowl paralysis, have given some indication of the progress which might be made in reducing loss from other diseases of poultry by the establishment of disease-resistant strains.

Deficiency diseases are outside the scope of this paper, but it should not be forgotten that a low plane of nutrition frequently lowers resistance to disease in individuals thus permitting a more rapid build up of infection leading to an outbreak which may have disastrous consequences in a flock of laying fowls, broiler chickens or fattening turkeys kept under the intensive conditions prevalent to-day.

Inadequate or faulty ventilation of intensive houses often proves to be the major predisposing factor in outbreaks of persistent respiratory disease among broiler chickens and deep litter laying flocks. Treatment with drugs or antibiotics under these circumstances is only temporarily beneficial and the long-term effect would be to add to the financial loss resulting from the outbreak.

It is essential that the veterinary surgeon in practice who wishes to extend his activities into the field of poultry should acquire for himself at least that amount of knowledge of poultry rearing and management that he would regard as necessary before he ventured to express an opinion on the cause of mortality or unthriftiness among cattle, sheep or pigs. When asked to express an opinion as to the cause of excessive mortality among baby chicks, turkey poults, or ducklings, the veterinarian should not forget that about 65 per cent. of the annual loss among poultry up to 4 weeks of age is due to non-specific factors, much of it the result of adverse environmental conditions, either at the hatchery, in transit to the customer, or during the first few days of rearing.

Although laboratory examination is necessary to eliminate the possible presence of a specific disease in baby chicks, a careful survey of the environmental conditions and circumstances under which baby chick mortality has occurred will often enable the experienced observer to offer helpful advice. Bacteriological examinations are, however, essential in the investigation of baby chick mortality, as quite apart from the risk of a mistaken diagnosis, laboratory confirmation is required by most hatcheries in support of claims for compensation from the owner in respect of losses occurring from disease

The specific bacterial and viral diseases, excluding leucosis and fowl paralysis, diagnosed during 1959 at Weybridge, in carcases sent for *post-mortem* examination represented only 14.0 per cent. of the adult birds examined, 15.2 per cent. of the growing birds, and 19.2 per cent. of the batches of chicks. Gordon (1959) in his paper to this Congress last year attempted to estimate the monetary loss represented by the figures available on mortality as they affect the broiler industry. He quoted a figure varying from £5 to £15 on a crop of 1,000 broilers, depending on the age at which the mortality occurred, and he referred to estimates made by the British Broilers Growers' Association indicating that a broiler producer with 8 per cent. mortality in the last week or so before killing would only just cover his costs of production, while if the figure rose to 12 per cent. a loss would be incurred. When stressing the effect of sub-clinical disease on cost of production, Gordon quoted a case where the percentage of second grade carcases rose from the usual level of 10 per cent. to about 30 per cent., resulting in a financial loss on that particular batch.

Unfortunately, there are no figures available concerning the corresponding financial loss in other branches of the poultry industry, but quite recently Gordon (1960) using statistics from laying trials and economic surveys estimated that disease caused an annual loss to the industry of at least £20 million.

Diseases of the Young and Growing Bird

Baby Chick (day old to 4 weeks old) mortality

Salmonella Infections

Salmonella disease is still the most important of the infectious conditions to which the baby chick is susceptible. Until the eradication of *S. pullorum* from our breeding flocks has been achieved by the application of the agglutination test for the detection of the carrier hen, there will always be a risk of mortality among baby chicks from this disease during the first fortnight or so of their life. Pullorum disease was diagnosed in 4.2 per cent. of the 3,856 batches of chicks examined at Weybridge in 1959, whilst infection by other Salmonella organisms, principally *S. typhimurium* and *S. thompson* was found in another 4 per cent. The economic significance of these figures is much less than it would have been 5 years ago before the availability of furazolidone for the prevention and treatment of these diseases. Many proprietary chick mashes contain the drug at the 0.01 per cent. level, which is often sufficient to prevent the serious mortality which would otherwise occur; if, however, infection is known to exist in a batch of chicks or turkey poults, 0.04 per cent. is necessary to check increased mortality.

When advising a client on the possible causes of excessive baby chick mortality during the first week or so, the veterinarian must first satisfy himself that the trouble is not due to environmental factors. Mismanagement in one of its various forms does not usually produce diagnostic lesions in the internal organs of dead chicks; neither are characteristic changes present in all outbreaks of disease; hence the need for routine bacteriological examination. If sufficient carcases are examined, however, certain changes can usually be detected, which will often justify a tentative diagnosis and appropriate treatment pending the results of a laboratory examination.

The chief lesions in baby chicks or turkey poults dead from *Salmonella* disease are enlarged mottled livers and spleens, myocardial necrosis, necrotic lung lesions and caseous caecal casts. The early recognition of the possible significance of these lesions, followed by a recommendation to use furazolidone in the mash may save the client a considerable sum of money. It must not be forgotten, however, that

treatment of *Salmonella* infection almost invariably leads to a higher carrier rate among the survivors, and that these survivors should never be retained as potential breeding stock.

The work of Carnaghan and Sojka (1958) on the occurrence of chronic pullorum arthritis among the survivors of an outbreak of this disease is a further indication that this condition still represents a threat to the profitability of a batch of broilers in spite of the effective action of furazolidone in reducing actual mortality during the acute stages of the disease.

Outbreaks of fowl typhoid in baby chicks are rare, but egg transmission of infection does occur, and outbreaks are encountered occasionally, particularly in those parts of the country where the normal incidence of fowl typhoid is high. The lesions of the disease are somewhat similar to those of other *Salmonella* infections, although once again confirmation of diagnosis is dependent upon the isolation of *S. gallinarum*, the causal organism, from the organs of dead chicks. Furazolidone controls this disease effectively, but a recent experience at Weybridge suggests that under broiler conditions the infection may flare up again before the affected batch has been killed, the organism no longer proving susceptible to drug treatment.

Coli Septicaemia

Under normal broiler conditions 1 to 2 per cent. of most batches of chicks die during the first week or so, due to the various non-specific factors already mentioned. About the same number of casualties occur during the remaining 9 weeks prior to killing. An increase in this relatively insignificant rate of loss is not uncommon after about the sixth week, often accompanied by slight respiratory symptoms. In some outbreaks the losses amount to 20 or so per day in batches of 8,000 to 10,000 chickens. *Post-mortem* examination of the dead birds reveals extensive purulent pericarditis with a fibrinous exudate over the surface of the liver and gross thickening of the abdominal air sacs, with an accumulation of caseous material; the liver is dark and congested and the spleen enlarged. As the name implies, this condition is the result of a septicaemic infection with certain strains of *Escherichia coli*. Diagnosis of the disease is confirmed on bacteriological examination by the isolation of these organisms in pure culture from the internal organs of the dead birds.

The majority of strains of *E. coli* isolated from outbreaks of this disease, prove to be susceptible to furazolidone and 0.04 per cent. of the drug in the food is the treatment usually recommended, but strains vary in their susceptibility to this drug and it is now routine practice to carry out antibiotic and furazolidone sensitivity tests on all cultures isolated from outbreaks, the appropriate treatment being recommended in accordance with the results of the test.

Recent experience at Weybridge suggests that the routine use of furazolidone at low levels in baby chick mashes for the prevention of *Salmonella* infection may result in the subsequent development of strains of *E. coli* resistant to this drug, a matter for concern should it become necessary to resort to treatment of an outbreak of coli septicaemia at a later stage.

Mortality in a broiler crop which has experienced an outbreak of coli septicaemia may reach 10 per cent. or more, but it is the increase in the number of second quality carcases which makes this disease a serious economic threat. It is, however, important that the veterinarian called upon to advise on the control and prevention of this condition should bear in mind the important part played in many outbreaks by adverse environmental conditions, such as faulty ventilation and design of houses, or overcrowding. Continued resort to drugs and antibiotics under such circumstances is at the best merely a palliative measure and owners should be urged to do everything possible to correct the underlying predisposing causes of the outbreak.

Infectious avian encephalomyelitis (epidemic tremor)

A description of the diagnosis and epidemiology of this virus disease of baby chicks as it occurs in this country was published by Markson and Blaxland in 1958. There is little new to add to this account of the disease. The economic loss from the condition is not high but the nuisance value is considerable to hatcheries and other suppliers of day old chicks. The symptoms are not always easily distinguished from those of other conditions causing leg weakness and inco-ordination, and although it is generally admitted that the disease is egg transmitted many commercial hatcheries insist upon the presentation of a laboratory report in support of claims for compensation.

There has been a tendency in recent months for clinical evidence of infection to develop in chicks at an earlier age than was previously the case, and it is not uncommon to encounter the disease in chicks only a few days old. Similarly, the well-marked seasonal incidence which was so characteristic a feature of the disease in the past is no longer apparent; this change, however, is explained by the virtual absence of a regular hatching season, the demands of the broiler industry now requiring a continuous supply of chicks all the year round. Investigation of outbreaks of I.A.E. still indicate that it is predominantly pullet flocks which are the origin of the disease. Dissemination of the virus in the eggs from an infected flock is of only a few days' duration, and infection is seldom traced to the same flock for more than one or two consecutive hatches. Nothing is known of the spread of the disease among adult fowls, although recent American reports (Schaaf, 1958; Calnek & Jehnick, 1959; Luginbuhl, Sumner & Jungherr, 1959) suggest that both adults and chicks which have survived an outbreak are immune to reinfection. Antibody has been detected in the serum of recovered fowls, and experiments have shown that a degree of protection is conferred by such parents to their progeny.

There is no treatment of any value in this disease and chicks showing symptoms are best culled. Work at Weybridge, using affected survivors of outbreaks of I.A.E. as parent stock has never suggested transmission of virus from parent to progeny, although

microscopical examination of the brains of the chicks revealed histological changes in approximately 8 per cent. in one experiment and 38.8 per cent. in another group (Markson & Blaxland, 1958) similar to those noted in field cases of the disease. For this reason it was judged unwise to retain the survivors of an outbreak of I.A.E. as potential breeding stock, even though they show no clinical evidence of the disease. It is possible, however, that this advice may have to be reviewed in future; it could be that clinically normal survivors of an outbreak of this disease may be the very birds most suitable for breeding purposes, as they themselves are immune to infection and may transmit protective antibody to their progeny.

Infectious Synovitis

This virus disease, first reported in Great Britain by Carnaghan (1959) tends to remain localised among broiler chickens and breeding flocks in certain areas.

The disease is seldom detected in chickens under about 6 weeks of age. The lesions of the disease on the feet, legs, hocks and wings are usually characteristic, but bacteriological examinations are necessary to differentiate this condition from the somewhat similar lesions caused by staphylococcal infection; occasionally outbreaks of synovitis are complicated in individual birds by secondary bacterial infection, thus making diagnosis difficult when based on the examination of only a small number of specimens.

It is now generally accepted that this disease is egg transmitted.

Fortunately a considerable measure of control is obtained over this infection by the feeding of high levels of antibiotics in the food. Both chlortetracycline and oxytetracycline are effective in holding the disease in check, but neither substance is viricidal and an affected flock of broilers has to be treated until a few days prior to slaughter if the development of further cases is to be prevented. Antibiotics are superior to furazolidone for the treatment of this disease and most workers have found that chlortetracycline is slightly more effective than oxytetracycline. (Shelton, *et al.* 1958; Snoeyenbos *et al.* 1958; Shelton & Olson, 1959). Although 200 g. of antibiotic per ton of food is the usual recommendation for the treatment of outbreaks of this disease, lower levels are probably effective if fed to an infected group of chickens from 2 to 3 weeks of age, as may be necessary when past experience suggests that the disease may be present in the progeny of a particular breeding flock.

The cost of treatment, which can be considerable when large numbers are involved, may be less in the future if commercial use can be made of the potentiation of tetracycline antibiotics by the addition of terephthalic acid which has been demonstrated experimentally by Price & Zollis (1959) and Cover *et al.* (1959).

Duck Diseases

A paper on the infectious diseases of young poultry would be incomplete without reference to 2 diseases causing mortality among ducklings, even though specialised table duckling production is largely confined to East Anglia. Veterinary surgeons in practice in other parts of the country should, however, be aware of these 2 potential causes of loss to those of their clients whose interests may extend to the production and rearing of ducklings.

Virus Hepatitis

Virus hepatitis is a highly infectious disease of young ducklings. It is characterised by its sudden onset and rapid violent course. Mortality of up to 90 per cent. may occur within 2 days of the first death in ducklings of under 3 weeks of age. Resistance to the disease increases rapidly with age, so that losses in ducklings infected at 4 weeks of age seldom exceed 5 per cent., and deaths from virus hepatitis are rare in ducks over 5 weeks of age.

Anatipestifer Infection

This disease is caused by a bacterial organism known as *Moraxella anatipestifer*. It usually commences in growing ducklings of 4 to 9 weeks of age, but may spread back to ducklings of brooder age and to older groups. It may occur as a severe spreading condition affecting almost all ducklings in susceptible groups, causing mortality of up to 75 per cent. In other flocks it causes a milder disease with lower mortality while occasionally it may occur as a sporadic condition affecting a few members of the flock only.

The diagnosis of these 2 diseases requires laboratory facilities, and it is not suggested that the veterinarian should attempt to diagnose them without laboratory guidance.

Asplin (1958) has described the use of attenuated strains of duck hepatitis virus for the passive protection of ducklings by inoculation of the breeding duck prior to collection of eggs for hatching and the active immunisation of young ducklings by foot web injection of virus at day old. This vaccine has been used experimentally in Norfolk under Ministry supervision, but it is not yet available commercially.

In outbreaks of anatipestifer infection, Asplin (1960) recommends chlortetracycline given in the food at the rate of 100 g. per ton for the prevention of fresh cases but the disease tends to recur if treatment is stopped.

Diseases of the Adult Bird

The number of infectious bacterial and virus diseases occurring to-day in our poultry flocks is too numerous to permit discussion of them all in detail in this paper, but the veterinarian who intends to advise his poultry-owning client should be familiar with the salient features of the more common of these diseases.

Virus diseases of adult poultry are chiefly those usually referred to as respiratory diseases. With the exception of chronic respiratory disease (C.R.D.) all the more serious respiratory diseases of poultry are viral in origin. The 4 major diseases in this group are Newcastle disease (fowl pest), infectious laryngotracheitis (I.L.T.), fowl pox, and infectious bronchitis. These conditions, together with C.R.D.,

were discussed by Chu in his paper to this Congress in 1958 and also by Gordon (1956). It is recommended that these papers be read again in conjunction with the present paper by those wishing to cover the whole field of infectious disease in poultry. Another useful Congress paper, is that by Blount (1955) on poultry therapeutics.

In the present paper, it is intended to discuss briefly some of the more important bacterial diseases affecting the older bird, drawing attention to any recent developments which have taken place, with particular reference to those conditions which the veterinarian in general practice is likely to meet.

(1) Bacterial diseases

Fowl Typhoid

Two recent developments of importance affecting treatment and control of fowl typhoid have been the use of furazolidone to combat actual mortality in an outbreak and the production of a live vaccine prepared from a rough strain of *S. gallinarum* for the immunisation of flocks.

Furazolidone (0.04 per cent. in the mash) can usually be relied upon to check the mortality following a diagnosis of fowl typhoid. Unless the flock is moved to fresh quarters after treatment, however, disease is almost certain to reappear after a short interval, and further treatment will be necessary. If treatment is delayed and disease has become well established in a flock, the results are less satisfactory, and a higher proportion of the survivors will continue to excrete the organism. Furazolidone treatment is of most value in combating fowl typhoid in commercial egg producing flocks. In breeding flocks, the survivors should always be blood tested after the conclusion of the first period of treatment and eggs should not be collected for hatching until 2 clean tests have been obtained at intervals of 2 to 3 weeks, subsequent to the outbreak. The agglutination test for the detection of carriers of fowl typhoid is similar to that for pullorum disease, and the same antigen detects carriers of both diseases. The need for precautions against the reintroduction of infection is even greater following an outbreak of typhoid in a breeding flock, and reactors to the blood test will be found continually unless all possibility of re-infection is eliminated. Infected houses, ground and equipment constitute the biggest risks but it should be remembered that other poultry and wild birds may constitute a potential source of infection (Harbourne, 1955).

Vaccine for immunisation of fowls against fowl typhoid is now available from the Animal Health Trust, Biological Products Department, Kennet, near Newmarket. Although the vaccine consists of a live organism, the strain used is avirulent, even to young chickens, but vaccinated birds do sometimes react to the agglutination test to a slight degree (Gordon, Garside & Tucker, 1959). The immunity conferred by vaccination lasts for from 4 to 6 months. In areas where fowl typhoid is widespread, all young stock should be vaccinated when they are about 8 weeks old, and until more information is available on the length of immunity following vaccination it is advisable to re-vaccinate older birds every year. When vaccine is used following an outbreak, survivors should not be vaccinated within about a week of the completion of furazolidone treatment, as there is some evidence to suggest that the drug may destroy the vaccine strain and thus prevent the development of immunity (Gordon, 1960).

It is felt that until more evidence is available concerning the behaviour of the vaccinal strain of *S. gallinarum* in a vaccinated flock, and in particular of its effect on the agglutination test (Gordon & Luke, 1959), it is unwise to resort to vaccination for the control of fowl typhoid in breeding flocks subject to routine blood testing for the prevention of pullorum disease. Vaccination is not normally permitted in flocks controlled by the Poultry Stock Improvement Plan of the Ministry.

Pasteurellosis

Classical outbreaks of fowl cholera (*Pasteurella aviseptica* infection) are extremely rare in this country, but a less acute disease with mortality varying in severity from individual birds to outbreaks with several deaths every day, are not uncommon in fowls and turkeys. In its chronic form the infection is often known as wattle cholera owing to the characteristic swelling of the wattles and intermaxillary space. *Past. aviseptica* is also frequently isolated from the upper respiratory passages and lungs of individual fowls on farms where respiratory diseases are a problem, and it is not uncommon in various species of wild birds (Keymer, 1958).

The organism isolated from acute and chronic outbreaks of pasteurellosis and from individual carriers appears to be identical to that causing the classical peracute disease (fowl cholera), except for its pathogenicity. It is rare in this country to isolate a strain of this organism which will kill a healthy laboratory fowl or turkey, even though mortality from that strain is apparently occurring in the field outbreak.

Diagnosis of this disease is dependent upon laboratory examination but the practising veterinarian may be called upon to advise treatment for a flock of fowls or turkeys in which this disease has occurred. Sulphaquinoxaline in the drinking water, and antibiotics at levels of 100 to 200 g. per ton in the food have been recommended. Intramuscular injections of oxytetracycline in oil (25 mg. per kg. bodyweight) are sometimes effective, and in a recent outbreak in turkeys Dwyer (1959) claimed good results following intramuscular injection of 0.5 c.c. aqueous suspension of chloramphenicol*. Treatment of this disease, however, can prove costly if large numbers of birds are involved, and the results are often disappointing, disease being liable to recur on the same premises at intervals†.

* Chloromycetin.

† Many attempts at control by vaccination have been reported but the results appear to have been disappointing in most instances. Recently Heddlestone and Reisinger (1959) described encouraging experimental work using a formalin killed organism.

Pasteurella pseudo-tuberculosis infection is not infrequently the cause of mortality among turkeys. This organism is commonly isolated from individual fowls, turkeys and also from wild and game birds (Keymer, 1958). Outbreaks among turkeys are usually associated with adverse environmental conditions, particularly if there is any overcrowding or during spells of cold wet weather. Treatment is even less satisfactory than with outbreaks of *Past. aviseptica* infection, and once again there are no specific remedies which can be relied upon to check the disease. High levels of antibiotics in the food, however, often seem beneficial, particularly if the build-up of infection has been associated with additional environmental stress.

Erysipelas

Mortality among adult turkeys due to *Erysipelothrix rhusiopathiae* infection is not uncommon. The swollen snoods of the stag birds and virtual absence of disease among hens is characteristic of this disease, but once again confirmation is dependent upon bacteriological examinations. Outbreaks are most frequent in the autumn months when the accumulation of stock for the Christmas trade leads to a degree of overcrowding, and losses frequently cease when flocks are divided up and allowed more space. Fighting among stag birds and consequent infection of small wounds and abrasions is one of the main factors leading to outbreaks. Treatment of individuals birds is seldom satisfactory owing to the difficulty of identifying the infected individual before the disease is advanced. 100,000 to 200,000 units of penicillin combined with 100 to 200 mg. of streptomycin given intramuscularly is a frequent recommendation. Procaine penicillin in the water or the food is recommended by some American workers (Boyer & Browne, 1957). Immunisation of turkey flocks using aluminium hydroxide adsorbed vaccines would seem to be a more reasonable way of combating this disease. American reports are encouraging (Adler & Nilson, 1952; Jerstad & Johns, 1954) and in a small laboratory trial carried out at Weybridge recently a considerable degree of protection was obtained using one of these vaccines.

Veterinarians carrying out autopsies on dead turkeys should not forget the painful condition resulting from accidental infection by *E. rhusiopathiae* and they should warn their clients of the risk to which they expose themselves through the careless handling of turkey carcases infected with this organism.

Staphylococcal Infection

Staphylococcal arthritis is a common cause of lameness, swollen joints, loss of appetite and debility in fowls, turkeys and ducks. Infection occurs at all ages and is frequent in the growing stages when it has to be distinguished from infectious synovitis and other diseases causing leg weakness. In the older bird it is more common among males, possibly owing to the greater weight of the average individual. When occurring on the feet the lesion is known as bumble foot, but the more usual site of this disease is the hock joint and associated tendons. The organism involved in this condition is *Staphylococcus aureus*.

It is customary to say that this disease is probably the result of infection of small cuts, wounds and abrasions, and it may well be so in many outbreaks, particularly when they involve birds housed intensively in wire-floored units and cages. But the condition is also encountered in flocks where these predisposing causes are absent and there may be other factors concerned in its aetiology.

Under experimental conditions staphylococcal infections have been treated successfully with antibiotics (Williams Smith, 1954), but in practice treatment is seldom worth while and ailing individuals are best culled. The possibility of predisposing environmental factors should not be overlooked.

Acknowledgments.—The author wishes to acknowledge the help of his colleagues in the diagnosis section of the Poultry Department at Weybridge, and in particular Mr. B. S. Hanson, M.R.C.V.S., and Mr. I. F. Keymer, M.R.C.V.S., without whose willing co-operation in recent years much of the material, on which this paper is based, would not be available.

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(2) Leucosis and Fowl Paralysis

BY

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SUMMARY.—The economic importance of the poultry industry in relation to agriculture as a whole is discussed and the magnitude of the losses occasioned by leucosis and fowl paralysis is briefly reviewed.

The more salient points regarding the post-mortem *findings in the diagnosis of these diseases from the general practitioner's point of view are considered.*

PERHAPS it is symbolic of the topsy-turvy world we now live in that the poultry industry, ranking second in agriculture only to that of cattle in the United Kingdom, remains the Cinderella among our professional interests. That such difference prevails is difficult to understand, more especially in view of the heavy losses the industry bears as a result of disease. Coles (1955) estimated the cost of mortality due to disease at about £10,000,000 in an industry worth £150,000,000 in the United Kingdom. He further suggested that the overall cost, as a result of death and loss of potential income, may even amount to £30,000,000.

Working either in the field or in a routine diagnostic laboratory it is impossible always to differentiate, so far as gross lesions are concerned, between all the various types of tumours affecting our domestic animals. That the same may be said with regard to tumour conditions in poultry is, therefore, understandable; yet this is surely no reason why we, as veterinarians, who alone have a knowledge of poultry pathology, should not play a greater part in this important industry. In particular practitioners, conversant with husbandry and management on the farm, are able to observe symptoms in detail, and as a result are well qualified to carry out such investigations. It is well known that the diagnosis of some of the disease conditions within the compass of the title of this paper present little real difficulty.

How important are the leucoses group of diseases and fowl paralysis?

Collectively this group of diseases accounts for a greater loss in poultry than any other. Blaxland (1956) analysing results of *post-mortem* examination records for Weybridge and Lasswade, has shown that approximately 24 per cent. of all adult poultry specimens examined showed evidence of leucosis or lymphomatosis. He went on to say that "the figures for the Poultry Production and Progeny Trials in Lancashire are even more disturbing, since here the records are kept of mortality from day-old to the end of the first laying season, *i.e.* 18 months old. For the years 1950-53 leucosis accounted for 35·7, 27·6, 41·0 and 53·0 per cent. respectively of a total average mortality of 21·7 per cent.". Records of *post-mortem* examinations of grower-adult specimens at Stormont, Northern Ireland, have shown a somewhat higher figure for 1959 than that for Weybridge or Lasswade. Of the 2,410 diagnoses made as a result of examining 5,379 grower-adult specimens, 22·93 per cent. were of neural fowl paralysis. The leucoses, the purely visceral form of fowl paralysis, and other tumours including bleeding cysts, together accounted for 37·6 per cent. of the diagnoses. Thus the diseases under consideration plus other tumour conditions accounted for 60·6 per cent. of all diagnoses in the grower-adult group.

The cost of mortality resulting from these diseases has been variously estimated. Gordon (1954) put the figure at nearly £4,000,000. Blaxland (1956) has suggested that the loss may be near £7-8,000,000; while based on figures by Coles (1955) he considered that the total loss as a result of death and of potential output, could be in the region of £15,000,000. In Northern Ireland output of the poultry industry was valued at approximately £24,000,000 for 1957-58. On the basis that loss from death and potential output is of the order of 20 per cent. of the annual value of the industry, and assuming that the leucoses and fowl paralysis account on the average for 20 per cent. of the annual mortality, the total loss may be in the region of £950,000.

What is meant by the term visceral lymphomatosis?

In American literature the term is used to signify a neoplasia of cells of the lymphocytic series. Smith and Jones's *Veterinary Pathology* (1957) describes it as the most common avian neoplastic disease. The use of the term on this side of the Atlantic is unfortunately by no means so well defined. When visceral lesions were first associated with fowl paralysis in Great Britain, the term visceral lymphomatosis was adopted in view of the fact that the neural form of the disease was already known as neural lymphomatosis. Since then, however, the American influence has complicated the picture to such an extent that it is sometimes difficult to decide which meaning is now intended.

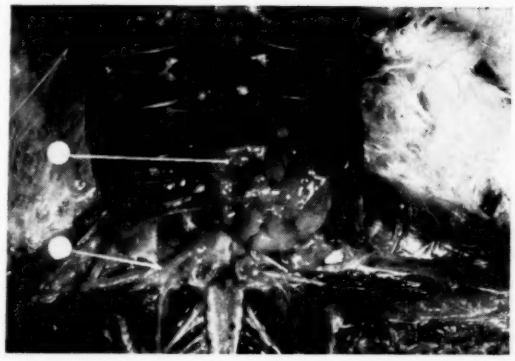


FIG. 1.—Ovarian lymphogranuloma and right femoral nerve.

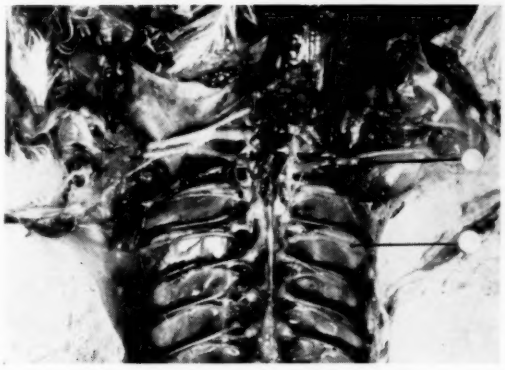


FIG. 2.—Gross changes brachial plexus and intercostal nerves. *N.B.* enlargement dorsal ganglia.

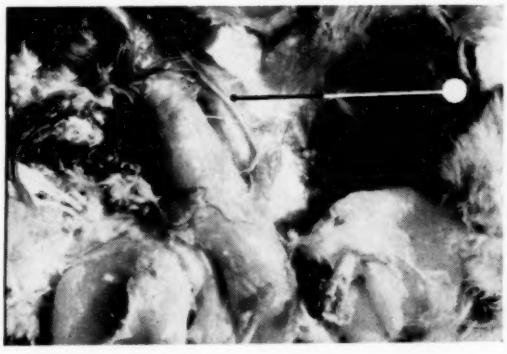


FIG. 3.—Marked enlargement left vagus nerve.

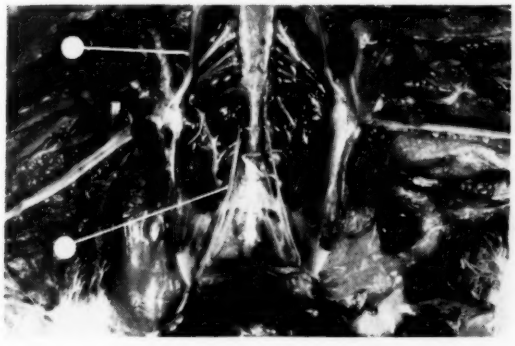


FIG. 4.—Obturator and unnamed pelvic nerves.

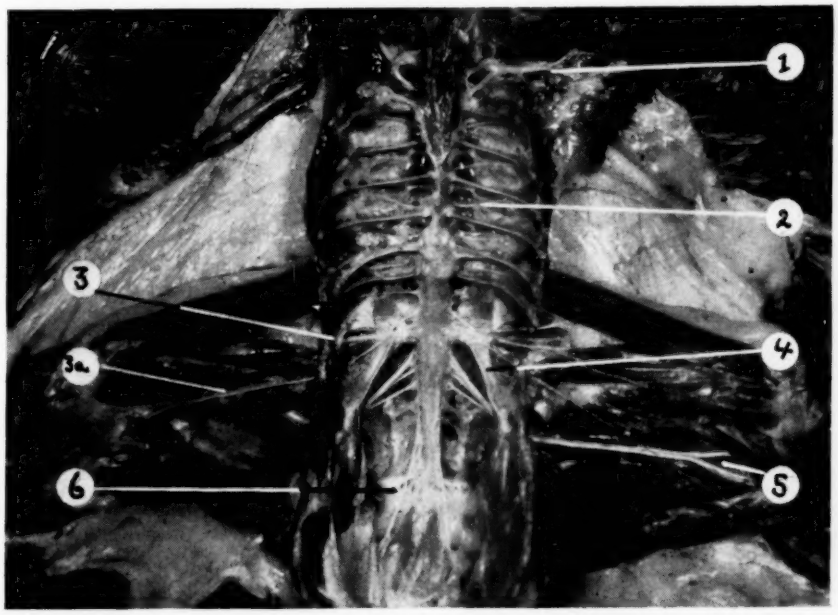
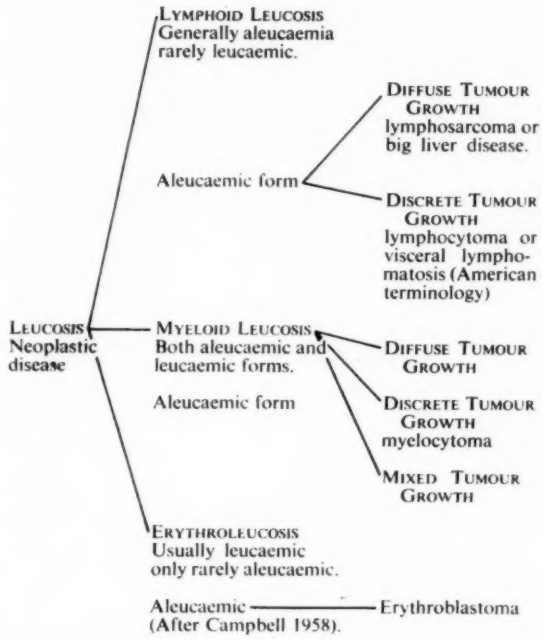


FIG. 5.—Normal specimen showing: 1, brachial plexus; 2, intercostals; 3, femoral (a) superficial branch of femoral; 4, obturator; 5, sciatic trunks; 6, unnamed pelvic nerves.

Are these diseases related?

For the purpose of this paper the diseases are discussed under the following headings: —



(After Campbell 1958).

Although it is customary to classify these diseases under a single heading such as avian leucosis, avian leucosis complex, or fowl paralysis complex, it was not originally intended by doing so to infer an aetiological relationship. It is apparent, however, that over the years such an inference has been widely made.

In Great Britain Campbell (1954, 1956) presented evidence for the separation of the leucoses from fowl paralysis. He affirmed that whereas the leucoses are neoplastic conditions affecting haemopoietic tissues, neural F.P. (neuro-lymphomatosis) is a chronic inflammatory condition and as such should not be included in the leucoses group. It is Campbell's opinion (private communication) that the visceral lesions of fowl paralysis or visceral lymphogranuloma, as he prefers to call it, are merely another manifestation of the neural form of the disease.

Leucoses

The term leucosis signifies a complexity of neoplastic conditions affecting the haematopoietic tissues, sometimes causing a true leukaemia. Three forms of the disease are described, lymphoid leucosis, myeloid leucosis, and erythroid leucosis.

Lymphoid leucosis is by far the most common of the 3 forms: it may occur either as a diffuse tumour growth (big liver disease) or as a discrete nodular tumour condition. The discrete form (Campbell, 1949) commonly affects the ovary, liver, spleen, kidneys, Bursa of Fabricius and the intestine, less commonly the lungs, heart and pancreas. Macroscopically the lesions may be confused with the visceral form of fowl paralysis and with other discrete tumour conditions. Campbell (1949) affirms that the ovarian changes resemble a carcinoma. The diffuse form of lymphoid leucosis causes gross enlargement of the liver, spleen, and frequently the kidneys. The affected organs, especially the liver, may weigh as much as a pound and a half compared with a normal liver which only weighs a couple of ounces. The liver, which is generally mottled, varies in colour from a creamy pink to a blue-grey, the surface being smooth. In many cases the changes are so marked that diagnosis is not a problem.

Myeloid leucosis occurs both as a discrete form (myelocytoma) and as a diffuse form. The discrete form is readily recognised as a friable chalky-white tumour condition mainly involving the surface of the sternum, the ribs and the pelvis. Lesions on the ribs, especially those in the costo-vertebral region, may be visible only after removal of the lungs. Similarly, it is necessary to dissect away the kidneys before pelvic lesions can always be seen. Sternal lesions, which may be seen on cutting away the keel or sternal crest, have been described by Campbell (1942). The liver presents a streaky effect, and the edges may be completely replaced by white friable tumour tissue. Other organs affected (Campbell, 1949) are the heart, lungs, spleen, ovary (testes) and kidneys.

The diffuse form of the disease described by Chubb and Gordon (1957) presents a marked enlargement of the liver and spleen, the liver having a granular or "Morocco-leather" appearance. Campbell (1949) describes the liver and spleen as greatly enlarged (the liver weighing as much as 400 g.), of firm consistency and having a finely mottled reddish-brown appearance with small white areas. The ovary, consisting of large rounded coalescing soft pink masses, may weigh 100 g.; it is apparent that myeloid leucosis may occur as a mixed tumour condition together with erythroleucosis (Campbell, 1949). It will be realised, therefore, that a diagnosis on macroscopic lesions alone is not always possible.

Erythroid leucosis. This form of leucosis and myeloid leucosis are known to be closely related. The erythroid form typically causes an enlargement of the liver and spleen, described by Chubb and Gordon (1957) as cherry-red in colour, soft and friable. Blood smears show large numbers of primitive red cells, the condition causing a true leukaemia. Death of the bird may result from rupture of the diseased spleen or liver.

Fowl Paralysis

In the past it has generally been assumed that F.P. was classically a disease of the near sexually mature bird. In recent years, however, the disease has manifested itself with increasing incidence in the younger bird. Jungherr (1959) alludes to it in birds 3 weeks of age. Our experience is that it is not uncommon in birds between 6 to 8 weeks and very

common in birds around 4 months of age. Chubb and Gordon (1957) in a survey of broiler losses found that 16 per cent. of the deaths were due to F.P., an incidence second only to caecal coccidiosis. So far as seasonable incidence is concerned, with the present more extensive hatching season and the fact that birds are exhibiting signs of the disease earlier in life, one need no longer expect the same seasonal mortality as described by Campbell (1956), *i.e.* from August to October. Wilson (1949) has discussed fowl paralysis extensively in Blount's *"Diseases of Poultry"* which is well worth reading. Three types of Fowl Paralysis are described: Neural, Visceral and Ocular.

Neural F.P. (*neurolymphomatosis*) may affect any of the peripheral nerves in the body. It is a mistaken idea merely to associate neural F.P. with a paresis of the legs or wings. Very commonly, apart from paralysis of the legs and wings, it affects the nerves to the neck, the crop, the respiratory system, the alimentary canal, the viscera or the tail, producing varying degrees of paralysis of the part involved such as torticollis, distended crop, respiratory or vocal embarrassment, digestive upsets, dropped wing, lameness or a dropped tail. The extent and variation of lameness exhibited by birds affected with neural F.P. are too numerous to recount. The text-book picture of the birds with clenched toes, one leg extended forward and the other backwards, although typical enough, is only one of a great many postures or symptoms exhibited. The *ante-mortem* signs of neural fowl paralysis, although suggestive, are not necessarily diagnostic, since paresis of the legs may be associated with conditions such as arthritis, perosis or vitamin B_2 deficiency.

Post-mortem examination: while a very casual, even superficial examination may show gross diagnostic changes in the more accessible nerves such as the brachial or sciatic plexuses, when such changes are not evident a much more exacting examination must be made if one is not to miss a high percentage of positive cases. When a superficial examination does not reveal evidence of the disease, a systematic search of all the accessible nerves should be made. First the vagal nerves should be compared and examined throughout their length, while at the same time one can observe the cervical nerves. The brachial plexuses are next examined, a comparison being made between the 2 sides. Particular attention should be paid to the origin of the branches where they emerge from the vertebral foramina, even when there may be no apparent changes in the nerve trunks themselves; one can diagnose neural F.P. on the changes in the dorsal ganglia. Sometimes the changes are so large that they can only be described as "tumoured". Campbell (1956) cited the occasional instances of such a conversion to a neoplastic growth. The intercostals should next be examined, again comparing the nerves on both sides. Attention should then be paid to the anterior coeliac nerve plexus, where occasionally the changes may be so extensive as to resemble a "tumour". The pelvic plexus supplying the more caudal portion of the alimentary canal, etc., may be seen in the mesentery alongside the rectum. The sciatic plexus consisting of the femoral, the obturator and the large sciatic trunks, along with ramifications of smaller unnamed nerves within the pelvis are next examined. In order to see the pelvic portion of the sciatic plexus, a portion of the kidneys must be removed. The extrapelvic portion of the sciatic trunk should then be inspected throughout its length by reflecting the triangular mass of the adductor muscles.

Pathology: The histopathological changes have been described in detail by Campbell (1956). Briefly, they commence as an oedema of the affected nerve followed by an infiltration of chronic inflammatory cells which may ultimately destroy all nerve structure. In the examination of specimens suspected of having fowl paralysis it is well to remember these histopathological changes, otherwise one may well be perplexed by the apparent absence of any visible nerve changes in the part affected, while visible changes may be evident in other nerves without apparent paralysis.

Visceral F.P.: Visceral lymphogranuloma, visceral lymphomatosis (non-American terminology). Campbell (1954) suggests that in about 10 per cent. of neural fowl paralysis cases visceral lesions may occur in the form of a tumour-like growth. The ovary is perhaps the most commonly affected organ, but lesions may occur in the liver, spleen, lungs, intestines and the Bursa of Fabricius. Muscles may also be involved, though less commonly. The affected ovary is obviously "tumoured" and may be as large as a small egg. It is usually pinkish-white in colour and although it is frequently described as brain-like in consistency, it is much firmer than brain tissue. The lesions in the other organs are discrete in type and often of a greyish-white colour. Involvement of the heart frequently causes marked distortion. When "tumour-like" growths occur in the absence of neural changes diagnosis is problematical. Campbell (1949) stated that both macroscopically and histologically it is frequently very difficult to distinguish between carcinoma and lymphoblastoma of the ovary.

Ocular Fowl Paralysis: Ocular lymphomatosis or "pearly eye", although occasionally occurring in a flock in which no other manifestation of fowl paralysis is evident, occurs much more frequently as a further complication of fowl paralysis.

Aetiology, Spread of Infection and Control

It is generally accepted that: —

(a) The causal agents of these diseases are viruses, although the infecting agent has not been isolated in the case of either lymphoid leucosis or fowl paralysis. Beard (1956) isolated the virus of myeloid leucosis in a pure form. It now seems likely that myeloid and erythroid leucosis are caused by separate agents.

(b) Adult carrier birds almost certainly play an important rôle in the spread of these infections.

(c) Chicks are particularly susceptible; indeed it seems extremely likely that the majority of birds become infected during their chick life. Birds from 10 to 16 weeks onwards generally show a reasonable degree of resistance.

(3) Coccidiosis

BY

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SUMMARY.—The biological characteristics of the life-cycles of four species of coccidia are considered in relation to the development of the associated diseases. The diseases, their economic importance and possible control are also discussed.

WHERE there is an exogenous stage in the life cycle of a parasite upon which it depends for its transmission from host to host the extent of parasitism within a host will naturally be related to the numbers of infective exogenous stages it picks up. The numbers of infective stages available to a host will depend upon those environmental conditions favourable to their longevity and development to infectivity. Moisture and temperature are the most important of these conditions in determining the extent to which the exogenous stages become infective. Oxygen is necessary also for the survival and development of these stages but under field conditions lack of it probably plays little part in exterminating coccidial populations. As coccidia pass through at least one, and usually more than one multiplicative cycle in the host, it follows that the denser the susceptible host population the greater the increase in the oocyst-population on the ground. As the number of hosts increases the wider will be the dissemination of oocysts. This is particularly true of early infections as the hosts will eventually develop degrees of resistance against the coccidia which will reduce oocyst-production. It often happens, however, that much damage has been done to a flock before an effective resistance has developed in its individual members. There is an extremely delicate balance between susceptibility and developing resistance to disease in the history of every flock.

More and more chickens are being raised intensively, the general picture of parasitism is changing and the associated problems of control are increasing. The reduction of floor space available to each chicken has now reached a limit which just permits its survival provided that it remains healthy. The limit has been exceeded as far as the practical control of

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Leucosis and Fowl Paralysis.—*Concluded.*

(d) The leucoses in general occur in sexually mature birds, while fowl paralysis commonly occurs in birds from 2½ to 3 months of age onwards.

(e) Spread of infection *via* the atmosphere may be all important. Cole and Hutt (1951) have shown that the mortality from lymphomatosis (American terminology) was nearly 4 times greater in birds separated by some 40 ft. from adult stock compared with those 100 ft. away.

Spread may also occur by other routes, such as indirectly *via* attendants, etc. With regard to egg transmission, Cottral *et al.* (1954) have shown that while certain forms of the leucotic group may be spread in this manner, it is not generally considered that egg transmission is of great importance. The transmission of fowl paralysis *via* the egg has never been consclusively proven (Cole & Hutt, 1951).

(f) The state of resistance or susceptibility is more evident in certain strains than in flocks. Unfortunately the application of this knowledge is not of great practical use so far as the average sized breeding unit is concerned, but it has obvious application in the larger breeding units.

(g) Treatment is useless, none of the drugs at present available is of any value either as treatment or as a preventive.

(h) A flock affected with some other disease during its rearing period is more likely eventually to show an incidence of F.P. than a flock reared without any such upset.

In conclusion, with regard to osteopetrosis, a disease frequently included in the disease complex under consideration, it is now evident as a result of works by Holmes (1958-59) that osteopetrosis is a separate viral disease which should no longer be considered along with these other diseases.

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disease by husbandry adjustments is concerned. Not only has the control of coccidiosis become more complicated but species which were at one time thought to be of negligible importance have now become serious threats to poultry health. The lot of the research worker is not an enviable one as his services are now being sought to unravel the knotty problems which the poultry farmer has created for himself. The research worker is now in much the same position as the statistician who is called upon to interpret and, if possible to make sense of, the results of a thoughtlessly designed experiment which he had no hand in designing.

The Life-Cycle of the Coccidia

The Oocyst

The life-cycle of all poultry coccidia includes 2 exogenous stages, the unsporulated non-infective oocyst and the sporulated infective oocyst. Sporulation is completed on the ground in 1 or 2 days when the temperature and moisture conditions are favourable. Sporulation time often varies from species to species and is used as additional evidence of their identity. The sporulation time will obviously play an important part in determining the rate at which birds become infected. Although each species has its own length of sporulation time at optimal temperatures it will be lengthened at lower temperatures until it is inhibited altogether at freezing point but will sporulate when the temperature of the environment rises. The extent of oocyst-production, which again differs from species to species, and the behaviour of the oocysts in different climates together with the length of the life-cycle in susceptible chickens all contribute to an understanding of the epidemiology of the diseases associated with the different species. Oocyst-production, which is of obvious importance, is determined by (1) the inherent ability of the species to produce oocysts in a susceptible host, the so-called reproductive potential of the species, (2) the extent to which resistance will develop in the host with consequent effect on oocyst-production and (3) the "crowding effect" in which oocyst-production is greatly reduced in heavy infestations. The reproductive potential may be defined as the extent of the ability of a species to produce oocysts, a quality which varies from one species to another. Roughly speaking, the resistance developing in a host increases with the numbers of sporulated oocysts ingested and therefore the small numbers of oocysts ingested at one time will produce more oocysts than large numbers. The "crowding effect" of heavy infestation cannot be fully explained. Brackett and Bliznick (1952) point out that extensive tissue damage and caecal core formation may interfere with the discharge or even the formation of oocysts of *Eimeria tenella*. These authors suggest that either the coccidia may produce a toxin which retards their development, or that an early stage of the life-cycle stimulate a host reaction which affects subsequent oocyst-production by other generations of parasites. The more oocysts taken in by the chicken the greater is the amount of antigen produced by the early intracellular stages of the life-cycle. It is now known that resistance to *E. tenella* is stimulated in the host by the asexual stages of its cycle which culminates in the large potentially pathogenic second generation schizonts.

There are in Britain at least 4 species of coccidia which are capable of causing disease in fowls and it is interesting to compare the incidence of these diseases in fowls of different ages in the light of the reproductive potential of the species concerned. *E. tenella* has a much higher reproductive potential than the intestinal species *E. necatrix*. *E. maxima*, another intestinal species, has the lowest reproductive potential of all the poultry coccidia. Brackett and Bliznick suggest that although reproductive potential is important in determining the nature of an epidemic the over-all number of oocysts an infection can produce under any conditions is probably at least as important as the maximum reproductive potential under certain limited conditions. *E. tenella* oocysts quickly accumulate on the litter because of the high reproductive potential and maximum total of oocyst-productivity of this species. For these reasons, as well as the virulent nature of the species, caecal coccidiosis is characteristically a disease of young chickens up to 10 weeks of age although equally severe disease can be induced in older birds which have not previously been exposed to infections of *E. tenella*. There appears to be no marked age-immunity in the chickens, the resistance commonly encountered in older birds having developed from earlier exposures to the parasite. *E. necatrix* is just as virulent as *E. tenella* but on account of its low reproductive potential and maximum oocyst-productivity, accumulates more slowly on the litter and is characteristically a disease of older chickens. Chickens exposed to the slowly increasing numbers of oocysts of *E. necatrix* may develop substantial resistance to disease by the time dangerous numbers of oocysts have accumulated, whereas chickens may, and often do, become exposed to massive infestations of *E. tenella* before they have developed any marked resistance to caecal coccidiosis. *E. acervulina*, another intestinal species, has a reproductive potential and maximum oocyst-productivity which exceed those of the other species. This species despite its enormous oocyst-output, rarely causes disease in young chickens or the deaths of older ones, although deaths can be obtained under experimental conditions by feeding millions of oocysts to susceptible birds. *E. acervulina* is becoming increasingly important as a pathogen of growing stock. This species is obviously much less virulent than *E. tenella* and *E. necatrix* from which it differs by its much more superficial parasitism which is restricted to the epithelial cells of the intestinal wall. The later asexual stages of *E. tenella* and *E. necatrix* occur in the sub-mucosa and severe infections cause widespread destruction of the tissues.

The Asexual Phase (schizogony)

This phase is particularly interesting as in *E. tenella* and *E. necatrix* it includes the stages that produce the recognisable lesions of the diseases. There may be several generations of schizogony each of which is a multiplicative stage. The asexual phase also stimulates the development of resistance in the

host. The mechanism by which this resistance is developed is not yet understood although some proof of its humoral nature has been obtained (Burns & Challey, 1959; Horton-Smith *et al.*, 1960) and evidence of circulating antibody has been obtained from work now proceeding at Houghton. In *E. acervulina* both schizonts and gametocytes may combine to cause disruption of the intestinal epithelium.

The Sexual Phase (gametogony)

The products of the male and female gametocytes fuse and produce oocysts. In cases in which relatively few schizonts are formed the gametocytes appear to be the pathogenic stage. For example autopsies of poultry which have shown symptoms of disease resulting from experimental infections of *E. maxima* in chickens or *E. adenoeides* in turkeys reveal large numbers of gametocytes and oocysts in contrast to *E. necatrix* or *E. tenella* infections which show large numbers of second generation schizonts. This raises an interesting point which has not yet been adequately considered. Are there too few schizonts in the *E. maxima* and *E. adenoeides* cycle to stimulate a recognisable resistance to further infection in the host? A marked resistance is developed to both species and it may be that when only small amounts of schizont antigen are available, as would be expected in these species, the far larger number of gametocytes may be responsible for the stimulation of the immune response. The gametocytes of *E. tenella* on the other hand are only weakly antigenic.

The Completion of the Life-cycle

The prepatent period starts from the ingestion of a sporulated oocyst and continues until a new crop of oocysts derived from this oocyst first appears in the faeces. This prepatent period lasts 7 days in *E. tenella* and *E. necatrix*, 4 days in *E. acervulina*, 6 days in *E. maxima* and under 5 days in *E. adenoeides*.

Summary of the Factors Conditioning the Epidemiology of the Coccidioses

The inherent virulence of the species and the relative susceptibility of the chickens are inseparable factors in producing disease. Epidemics arise as a consequence of mass infestation of susceptible chickens by organisms of varying virulence. There is a lower limit of oocyst-intake at one time, or over a period of time, which is able to promote absolute resistance in the host and these limits again vary with the species of coccidia. The time that epidemics can be expected in groups of susceptible chickens is related to the virulence, reproductive potential and maximum oocyst-production of the species of parasite concerned. Eventually, immunity will intervene to reduce or completely suppress oocyst-production resulting from reinfection. In the field most fowls appear to be only partially resistant to parasitism.

Most healthy fowls can carry one or more species of coccidia. Such fowls provide the source of infections which may prove dangerous to fully susceptible chickens. The life-cycle of a coccidium is self-limiting and the high incidence of coccidial parasitism in fowls at large indicates the wide distributions of coccidia in the field.

The Different Forms of Coccidiosis and their Diagnosis

Caecal Coccidiosis

The intracellular life-cycle of *E. tenella* is confined to the caeca and occasionally in heavy infestations overflows into that part of the main intestinal tract into which the caeca empty. In acute cases of caecal coccidiosis copious blood is passed with the faeces. Examination of the caeca at autopsy reveals haemorrhagic patches which are visible through the caecal walls and which mark the sites of parasitism by the numerous second generation schizonts. The lumina of the caeca are filled with blood. Blood originating from the infection of the small intestine by *E. necatrix* may sometimes be drawn into the caeca, a condition which can easily be confused with an *E. tenella* infection. In this case, however, no macroscopic haemorrhagic foci will be seen through the caecal walls.

Intestinal Coccidiosis

1. *E. necatrix.* Part of the life-cycle of this species is passed in the intestine and part in the caeca. The faeces become blood-tinged and eventually contain haemorrhagic mucus and urates. The pathogenic colonies of large second schizonts occur in the "ballooned" small intestine and are marked by haemorrhagic and white spots which can be seen through the wall before the intestine is opened. Blood and blood clots are present in the lumen. Smears of the intestine wall and contents will reveal schizonts and merozoites but no gametocytes or oocysts will be found as gametogony is confined to the caeca.

2. *E. acervulina.* Heavy infestations with this species cause thickening of the intestinal wall. When the intestine is opened the walls may be inflamed and whitish areas which may be visible through the intestine wall will be seen on the surface of the mucosa. These tend to be obliterated in really heavy infestations by severe enteritis. Smears of the intestinal content will show large numbers of egg-shaped oocysts.

3. *E. maxima.* Heavy infestations with this species may cause a marked haemorrhagic enteritis and orange-coloured intestinal contents. Smears of the intestinal content will reveal the characteristically large orange-tinged oocysts of the species.

The Economic Importance of Coccidia

E. tenella is still a major cause of disease especially in warm wet weather. Caecal coccidiosis was once regarded as a seasonal disease occurring in the late spring and early summer. This is probably still true of the disease in chickens kept extensively, but in this age of broiler production and continuous production it has become an all the year round disease.

E. necatrix disease occurs at a later age than caecal coccidiosis and affects young growing stock often after the sixth week of age. Moderate infestations cause pronounced loss in weight and recovery is slow, hence the unfortunate tag "chronic coccidiosis," which is sometimes applied to the disease. Acute and fatal disease often develops but the epidemics are usually not as explosive as those of caecal coccidiosis.

E. acervulina may run a prolonged course. Severe infestations are marked by a reduction of feed intake, and the loss in bodyweight may be as much as 50 per cent. within a few days. This species is probably the chief cause of coccidiosis in laying birds in which egg production is affected.

E. maxima is the least important species from the economic angle although severe cases have been observed in the field and experimental infections have produced marked pathological changes but only rarely have they caused deaths. Under intensive conditions the species will probably increase in incidence and its associated disease in severity.

Control of the Coccidial Diseases

1. *By General Husbandry*

The importance of good husbandry cannot be overemphasised. Removal of infected faecal matter and litter before the oocysts have had time to become infective is sound in principle but under most modern conditions is quite impracticable. The behaviour of birds in a flock may indicate sites of heavy parasitism. Obviously the heavy parasite populations in the litter will be in those parts of the litter most frequented by the chicken. If there is a tendency for chickens to congregate on relatively small areas of litter it is important that the litter in such areas be removed at frequent intervals and replaced by new material. Re-distribution of the litter by raking will break up concentrations of oocysts and help to distribute them more thinly over the surface of the litter in such a way that chickens will pick up smaller numbers at one time. By picking up small numbers of oocysts regularly there is more likelihood of the chickens developing a resistance to subsequent reinfections. Coccidial oocysts are quickly destroyed by dryness and every effort must be made to keep the quarters as dry as possible. The litter can also be heaped between batches of chickens when the heat developed within its mass will rise to a temperature which is lethal to most stages of parasites. Water can be sprinkled over dry heaped litter to accelerate fermentation. There is a fire risk if the heap is in contact with wooden floors. Ammonia is still the most effective and practicable disinfectant to apply to surfaces of feeders, waterers and perches.

2. *By Chemical Means*

Broiler production has so complicated the whole question of control of coccidiosis by husbandry methods that various chemical substances have been introduced which are added to the food or water. Most broiler plants are using medicated feeds of one sort or another as prophylactics against coccidiosis. The early recognition of disease is essential if adequate control is to be obtained. In modern broiler plants it is almost impossible to pick out sick individuals or to see blood in the faeces and an epidemic may be well launched before the farmer recognises the disease. As broilers can be a paying proposition the farmer plays for safety and adopts some scheme of preventive medication which has become, whether we like it or not, a routine practice. A number of drugs used in this way have done a good job in reducing losses from caecal coccidiosis.

Recently, we have found strains within species which do not respond to the same kind of medication and there is also evidence that one kind of coccidiostat may not control more than one species. How widespread these strain differences are is still uncertain but their existence adds a further possible complication in selecting the form of medication to be adopted. We now have drugs which are equally effective against *E. tenella* and *E. necatrix* but may not be so effective in controlling *E. acervulina* and *E. maxima* which readily respond to sulphonamides.

Preventive medication seems to be justified in the case of broilers for the reasons given, but whether or not it is always desirable in older birds is doubtful. The suphonamide drugs can be relied upon to give satisfactory control of established infections of *E. tenella*, some strains of *E. necatrix*, *E. acervulina* and *E. maxima*. Where there is a history of recurrent outbreaks of intestinal or caecal coccidiosis a prophylactic scheme of treatment may have to be adopted. It may be unwise to use sulphonamides over long periods owing to side-effects. Appropriate prophylactic medication may have to be considered in which case it is of value to know the species of coccidium which is responsible for the disease and to prescribe the most effective drug against this species. It is possible that some of the newer anticoccidial compounds may prove to be effective prophylactics against all species.

There is ample evidence to show that it would be unwise to release drugs on to the British market which have only been tested against American strains of coccidia. These drugs must be tested in Britain to determine their effectiveness in inhibiting or suppressing the coccidial cycle, they must not interfere with the development of resistance and they must not produce ill-effects in the birds. In connexion with toxicity it is simply not enough for the parasitologist to record the effects of the drug on weight and to label the drug either as harmless or harmful according to whether weight gain is maintained or not. I believe that possible toxicity is too specialised a problem for an amateur toxicologist. When a drug has satisfied the requirements of an efficient coccidiostat it should be passed to a competent toxicologist for appraisal and approval before it is released for sale.

In controlling coccidiosis, it must be remembered that the flock is the patient and not the individual bird. The flock is a valuable unit which deserves all the care and attention that the veterinary surgeon, as an expert in disease, can give it.

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(4) Poultry Nutrition and Nutritional Diseases

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SUMMARY.—This paper represents an attempt by the author to give the veterinary surgeon in general practice a guide to present-day poultry nutrition and to some of the more common nutritional disorders which may arise. The introduction to the paper illustrates the economic importance of the poultry industry. The paper is then divided into sections as follows :—

General Nutritional Considerations.
Nutritional Diseases.
Abnormalities of Egg Shell Quality.
Abnormalities of Hatching Eggs.
Relationship between Nutrition and Disease.

Section II has been sub-divided into abnormalities of various systems or regions of the body. This method has been followed, as it was thought to be of more practical value than describing diseases associated with specific deficiencies or excesses which may not be seen in the field as separate entities.

Section V draws attention to the fact that as far as nutritional disease is concerned it is extremely difficult to recognise any clear-cut distinction between health and disease. Some relationships are quoted between the nutritional status of a bird and parasitic or bacterial disease. Finally, attention is drawn to the increasing importance of this subject.

IN a paper with a title covering such a wide field it is impossible to deal with the subject in complete detail. It has, therefore, been decided to attempt to give the veterinary surgeon in practice a general account of modern poultry nutrition and also some guidance as to the occurrence and aetiology of the nutritional disorders most likely to be met in practice. A short discussion on the subject of the relationship between nutrition and disease has also been included.

To-day, the poultry industry is reputed to be the second most important branch of agriculture. The number of poultry in this country has shown a remarkable increase over the last 10 years, which is borne out by the following Tables (Coles, 1960).

TABLE I
FOWL POPULATION (IN 000's) ENGLAND AND WALES, JUNE CENSUS, ADULT BIRDS AND TURKEYS

Year	Adult Birds	Turkeys
1950	28,731	753
1959 (Provisional) ...	35,066	2,308

TABLE II
ESTIMATED OUTPUT OF TABLE CHICKENS INCLUDING BROILERS IN ENGLAND AND WALES (MILLIONS)

Year	All Table Chickens	Broilers
1953-54	19	5
1959-60 (Estimated)...	108	100

It will be noticed that the biggest increase in numbers has taken place in the broiler industry and this increase is still continuing. Adult, presumably laying, stock have also increased while the turkey population has about trebled.

It is also of interest to note the comparative amounts of compensation paid by the Government in their attempts to control foot-and-mouth disease, tuberculosis and fowl pest in 1957. (Report on Animal Health Services in Great Britain, 1957.)

TABLE III
STOCK SLAUGHTERED AND COMPENSATION PAID

	Number	Value
Foot-and-mouth ...	30,410	£880,586
Bovine tuberculosis...	12,008	£658,727
Fowl pest	1,130,972	£1,115,598

As a further indication of the relative importance of the poultry industry the official estimates of compounded animal feeding-stuffs for the years 1953 and 1959 are listed.

TABLE IV
U.K. PRODUCTION OF COMPOUNDS

	1953 (Tons)	1959 (Tons)
Cattle...	1,788,304	2,883,700
Pig	1,567,148	1,502,300
Poultry	1,731,242	3,209,500
Others (Horse, sheep, etc.) ...	79,664	128,600
Total : ...	5,166,358	7,724,100

In recent years there has been a gradual change to more intensive methods of housing in an attempt to increase the productivity of poultry and decrease the production costs. As a result of this and the greater genetic potential of the birds, advances in nutrition have been stimulated. As food accounts for 70 to 80 per cent. of production costs it became increasingly essential to improve food efficiency in order to lower food costs and so increase profitability. The combined effect of factors such as breeding, feeding and management on egg production is indicated in the following Table (Coles, 1960):

TABLE V
ESTIMATED ANNUAL AVERAGE EGG PRODUCTION PER BIRD FOR ALL ADULT BIRDS ON AGRICULTURAL HOLDINGS IN ENGLAND AND WALES

Year	Egg Production
1950-51	145
1953-54	165
1958-59 (Estimated) ...	180

Section I: General Nutritional Considerations

As further knowledge is gained, it is becoming more evident that for the greatest efficiency the essential constituents of the diet must be related to each other as well as to the probable performance of the stock. When a practical ration is formulated a number of factors must be considered together, such as—

(*a*) The type of stock to be fed.

(*b*) What type of performance is required of the stock, *e.g.*, fast or slow growth?

(*c*) The ratio of digestible energy of the food to its protein content.

(*d*) If the ration has a high energy value, the relative amounts of vitamins and minerals may have to be increased, *e.g.*, modern high energy diets produce a type of physiological stress which increases the need for vitamin E in the diet to combat crazy chick disease (Biely & Marsh, 1953).

(*e*) What supplementary constituents should be added to ensure that the ration is complete and that the factors likely to limit the performance of the animal are eliminated? *E.g.*, is there enough of each essential amino-acid to ensure that there is no wastage of the other essential amino-acids already present in adequate amounts?

It is hoped that these examples serve to illustrate the complexity of modern poultry food formulation brought about by the necessity for more efficient feeding and the higher performance expected of the birds.

Water and Dry Food Intake

It need not be emphasised here that water may well be the most important part of an animal's diet, but it was thought desirable to make some mention of approximate water intakes of various classes of poultry due to the increasing number of drugs administered through the medium of water as a method of disease control. A convenient guide to the approximate daily food intake is also given:—

TABLE VI
APPROXIMATE DAILY CONSUMPTION PER 100 BIRDS

	Food	Water
Chicks (4 weeks) ...	7 lb.	1½ gal.
„ (8 „) ...	15 „	3 „
Growers (16 „) ...	25 „	5 „
Adult Fowl	30 „	6 „
Turkeys (4 weeks) ...	15 „	3 „
„ (8 „) ...	30 „	6 „
„ (16 „) ...	60 „	12 „
„ (24 „) ...	85 „	16 „
Adult Turkeys ...	100 „	20 „

Energy

The main sources of energy are fat and carbohydrates. In practical poultry rations cereals were relied upon to supply energy as carbohydrate and little attention was paid to the fats. However, owing to the reduction in price of edible fats and oils and the need to formulate high energy rations to promote more efficient and faster weight gains in broilers, fats are now incorporated in many commercial poultry foods. The effect of energy on the food intake and egg production of laying birds can clearly be seen in the following figures (Harms, 1959), which refer to a food calculated to contain 900 cals. P.E. per lb., compared with another containing 721 cals. P.E. per lb.

TABLE VII
EFFECT OF ENERGY ON FOOD INTAKE AND EGG PRODUCTION

	Egg Production	Food/Doz. Eggs
1954	+8%	−0.8 lb.
1955	+6%	−0.6 lb.

Protein

The protein content of a bird's diet must be considered quantitatively and qualitatively. Insufficient protein will reduce the rate of growth or production, but it is probably equally important to consider the amino-acid content of the protein and the relationship of the essential amino-acids to one another. The theory of limiting amino acids can best be illustrated by the analogy of a broken barrel.

If the diet contains the exact requirements for the essential amino-acids then they can be represented by the panels of barrel A—which are all of equal height.

Should one amino-acid be in excess of requirements it will not permit extra growth as illustrated in barrel B which, despite one panel being higher than all the others, cannot hold any more water than barrel A. Barrel C illustrates the fact that if one essential amino-acid is not present in sufficient quantity, then the diet will not support maximum growth even if another unrelated essential amino-acid is present in greater amounts than is necessary *i.e.*, the barrel can hold water only up to the top of the broken panel.

Thus, in any complete ration, the protein must be complete in essential amino-acids at the required level and in proper relationship to each other. Broadly speaking, the limiting amino-acids in poultry rations are likely to be methionine and/or cystine, lysine, glycine, arginine and tryphtophan.

Energy : Protein Ratio

As previously indicated, the efficiency or otherwise of a poultry ration can be attributed in some measure to the energy-protein relationship of that ration. Birds at various stages of growth or production require a certain amount of protein and, in order that some of this is not wasted, a sufficient amount of energy must be supplied. Conversely, if too much energy is supplied then some of it will be wasted due to the restriction of protein. If the energy:

protein ratio is too wide (*i.e.*, too much energy or too little protein) there will be a tendency to lay down fat, and if the ratio be too narrow there would be a tendency to excrete some of the protein which might lead to nephritis.

The energy: protein ratio may be expressed in either of three main methods:—

(*a*): Nutritive Ration (N.R.) which is the ratio of the digestible energy of carbohydrates and fats to the digestible energy of the protein and the urine. This can be expressed simply as digestible carbohydrate + 2.25 × digestible fat: digestible protein.

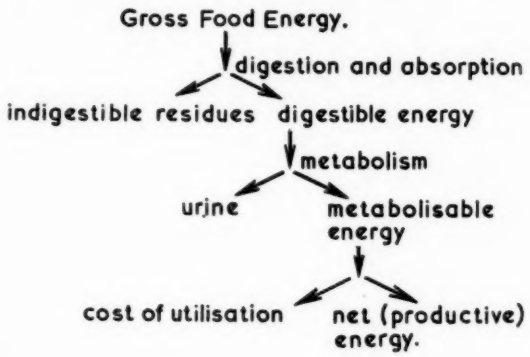
(*b*): Productive Energy: Crude Protein (PE: CP). Here the productive energy is defined as the energy in the food which can be stored by the animal as fat or protein. It represents the net energy of the food for production.

(*c*): Metabolisable Energy: Crude Protein (ME: CP) Metabolisable Energy is the total digestible energy in the food less that in the excreta and is therefore all the energy which can be utilised by the animal for fattening, production or maintenance.

The relationship between the different methods of measure energy values is shown:—

TABLE VIII

Utilisation of Food Energy.



It would seem that metabolisable energy is probably of most value in comparative energy studies for two main reasons:

(*a*): Digestible Energy does not take into account the value of the food to the animal—*i.e.*, variations occur in the ability of birds to utilise the food.

(*b*): Net (Productive) Energy does not take into account the varying requirements for maintenance and production—*i.e.*, rate of growth does not influence the energy value of the food.

However, at the present time, productive energy values are referred to most commonly. Energy may be measured as calories per 100 grammes, calories per lb., or therms per lb. The energy: protein ratio is also generally referred to as calories per protein or, more simply, C: P.

The following Table indicates in broad outline the energy and protein levels and C: P ratios which form the basis of most commercial poultry foods to-day, and should not be taken as hard and fast rules but as a guide for practical formulation (Hill, 1959).

TABLE IX

ESTIMATED ENERGY AND PROTEIN LEVELS FOR VARIOUS RATIONS

Type of Ration	ME: CP	PE: CP	Crude Protein
Chick (0-8 weeks) ...	65: 1	45: 1	19%
Grower (8-16 weeks)...	72: 1	50: 1	15%
Layer	81: 1	56: 1	16%
Broiler (0-6 weeks) ...	61: 1	42: 1	23%
Broiler (6-10 weeks)...	70: 1	49: 1	20%
Turkey (0-4 weeks) ...	43: 1	30: 1	28%
Turkey (4-8 weeks) ...	53: 1	36: 1	24%
Turkey (8-16 weeks)...	67: 1	46: 1	20%
Turkey (16-Killing) ...	93: 1	64: 1	15%

It will be noted that the figures reflect the type of growth or production required of the various classes of stock, *e.g.*, young stock have closer ratios, indicating higher protein content of diet, while fattening birds have wider ratios indicating the high energy content.

Minerals

In practical diets there are relatively few minerals which require particular and detailed discussion. There are numerous tables of suggested requirements for the different classes of stock—*e.g.*. recommendations by the Nutritional Research Council of the U.S.A. (1954), or in the Report of the Proceedings of the Brighton Conference (Scientific Principles of Feeding Farm Livestock, 1959). However, generally speaking, only calcium, phosphorus, manganese, zinc, iodine and salt are added to present day rations, as the requirements of other minerals are met by the main constituents of the diets. Selenium seems to be of some importance in the U.S.A.

Calcium

Levels of calcium in the diet of growing stock should be approximately 1 to 1.5 per cent., 2 per cent. for turkey poults and 1.75 to 2.5 per cent. in rations for laying stock. Some disagreement exists as to the amount of total calcium required by adult laying stock where 2.5 per cent. was thought to be sufficient, but recent work indicates that up to 3.75 per cent. (Peterson, 1959), might have a beneficial effect in egg-shell quality. There is considerable variation in the proportion of the total calcium requirement for laying stock included in present-day rations depending on the associated recommendations on supplementation. The laying bird will usually be given an allowance of a soluble calcium grit which enables the bird to meet its requirements for egg production.

Grit

The importance of grit to poultry is often not fully realised by the poultry farmer and 2 main types must be considered.

(a): Soluble grit (limestone grit, oyster shell) is basically a source of calcium for laying stock to balance their requirements for egg production and will not normally be a feature of the diets of growing stock.

Insufficient data of a critical nature is available about definite requirements of soluble grit but it should be regarded as a supplement to dietary calcium. Usually

about one quarter of the total calcium requirement is fed as soluble grit.

(b): Insoluble grit (granite or flint grit) should be supplied to aid the gizzard to perform its natural function of grinding the food. Approximate requirements are given.

TABLE X

APPROXIMATE REQUIREMENTS OF POULTRY FOR INSOLUBLE GRIT

Stock	Amount/100 birds/week	Size
Chicks (2-8 weeks) ...	1-5 oz.	Chick
Growers (8-16 weeks)...	6-8 „	Growers
Layers (Batteries) ...	8-12 „	Layers
Layers (Deep Litter) ...	ad lib.	Layers
Turkeys (2-10 weeks) ...	1-6 „	Chick & Growers
Turkeys (10 weeks—Killing)	8-10 „	Layers
Turkeys (Adult layers)	ad lib.	Layers

Phosphorus

It is not thought to be necessary to enter into a detailed discussion of the possible phosphorus requirements of the various types of stock nor into a description of the involved relationship between calcium, phosphorus and vitamin D. The most desirable ratio of calcium : phosphorus in the diet of growing chickens or turkey poults would seem to be between 1.5:1 and 2:1. Adult fowl and turkeys require a much wider ratio due to the high intake of calcium necessary for egg-shell formation and it would appear that phosphorus requirement for these birds is in the region of 0.8 per cent. of the diet, of which about half should be non-phytin phosphorus.

Manganese and Zinc

These elements are important for their inter-relationship with calcium and phosphorus metabolism and are generally included in commercial rations to provide levels of approximately 50 to 60 p.p.m., and 30 to 50 p.p.m., respectively. The requirement of turkeys is probably about 30 per cent. higher.

Iodine

May be important when rations are used which are low in animal protein. Less than 2 p.p.m. are required for normal thyroid function (Creech *et al.*, 1957).

Salt (Na Cl.)

Total levels of 0.5 to 0.75 per cent. are usually included in practical poultry diets.

Vitamins

It is thought to be of doubtful value in a paper of this nature to enter into a detailed discussion of recommended vitamin levels. In practical formulation, probably the most important vitamins are A, D, E, K, riboflavin, choline, pantothenic acid, nicotinic acid and B_{12}. It is likely that the latter 3 will not require to be added in synthetic form (except perhaps in high energy broiler rations or rations intended for adult breeding stock) and the levels of the others in Table XI are listed for reference purpose only and not to be taken as exact requirements.

Vitamins A and D_3 are now generally added to commercial poultry foods in a protected form, thus ensuring a longer life in mixed feeds. The vitamins are incorporated in small beadlets of microcrystalline wax or gelatine and this represents a significant advance over the inclusion of fish liver oils.

Unidentified Growth Factors

The factors include many supplements which, when added to foods, have improved the growth or performance of the animal, and they include the fish factor, whey factor, fermentation factor and the grass factor. There is some evidence to indicate that the whey and fermentation factors are the same (Wakelam, 1959). Practical rations generally include at least one of these factors and often a combination of two or three.

Other Additives

It has been decided to mention a few of the "additives" which may be included in present-day poultry foods. The order of discussion is not necessarily the order of their importance or merit.

(a) Antioxidants and their uses have become better known in nutrition during the last few years, probably due to the inclusion of fats in poultry rations, as obviously the fats and oils must be prevented from undergoing degeneration resulting in rancidity and unpalatability. Antioxidants can also aid in preventing the destruction of fat soluble vitamins.

(b) Bentonites are sometimes used to help the bonding of pellets during manufacture. They contain a fairly high proportion of silicates which is unlikely to have

TABLE XI

SUGGESTED VITAMIN CONTENT (PER LB. OF FOOD) OF VARIOUS RATIONS

	A(I.U.)*	D_3(I.C.U.)*	E(mg.)	K(mg.)	Riboflavin(mg.)	Choline(mg.)
Chick (0-8 weeks)	2,000	400	5-10	0.2	2.00	600
Growers (6-16 weeks)	2,000	400	„	„	2.00	600
Layers	2,000	400	„	„	2.00	600
Breeders	3,000	600	5-15	„	4.00	600
Broiler Starter	3,000	600	„	„	2.50	700
Broiler Finisher	2,000	400	„	„	2.00	600
Turkey (0-8 weeks)	3,000	600	„	„	2.50	700
Turkey (8-16 weeks)	3,000	600	„	„	2.50	700
Turkey (16 weeks-killing)	2,000	400	5-10	„	2.00	600

*If protected vitamins are used, half these amounts may be adequate.

any effect on the digestability of the food but Bentonite has been implicated in the destruction of unprotected vitamin D (Duckworth, 1959).

(c) Antibiotics as well as coccidiostats and anti-blackhead drugs etc., are often included in foods for disease prevention or even curative effects. In the case of antibiotics, such as chlortetracycline and oxytetracycline†, there is an undoubted growth response to low level inclusion rates (10 g.±5 g. per ton). In a recent paper it was suggested that antibiotics at this level may remove growth depression rather than cause growth promotion (Briggs, 1960).*

Continuous feeding of higher levels of antibiotics (25 g.±5 g. per ton) to adult laying stock have shown marked economic advantages where egg production is lower than 50 per cent. (Taylor, 1959).

Section II: Nutritional Diseases

It is the intention to discuss the more practical aspects of nutritional disorders rather than enter into a detailed account of theoretical deficiencies produced by the feeding of rations specially designed to be deficient in one mineral or vitamin. The main diseases associated with nutrition have been classified either systemically or regionally in the hope that this paper will have a greater appeal to the veterinary surgeon in practice.

Some indication of extent of nutritional disorders is given by Gibson and Harris (1960), who state that specific nutritional diseases are present in about 4 per cent. of the batches of fowls submitted to them for *post-mortem* examination. Non-specific conditions, such as impactions, are present in a further 5 to 8 per cent. They add that the figures for turkeys are very similar.

TABLE XII

ANALYSIS OF *Post-mortems* RELATED TO NUTRITIONAL DISEASES. 12 MONTHS 1958-59—VETERINARY INVESTIGATION CENTRE, NORWICH

	Chicks 0—28 days	Growers 1 month-P.O.L.	Adults	Total
Batches examined	412	604	675	1,691
Specific conditions				
Riboflavin deficiency	2	—	—	2
Encephalomalacia	15	19	0	34
Rickets/Osteomalacia	1	6	5	12
Perosis	1	4	—	5
Salt poisoning ...	1	—	—	1
Vit. A deficiency ...	—	4	15	19
			Total :	73 (4%)
Non-specific Conditions				
Impactions	4	51	28	83 (5%)
Digestive derangements ... ... (May include end result of intestinal coccidiosis)	4	37	22	63 (4%)
			Total	146 (9%)

While this is a local impression, it agrees fairly closely with the figures obtained from an analysis of *post-mortems* at veterinary investigation centres throughout the country in 1956 (Blaxland, 1958).

*Aureomycin.
†Terramycin.

General Consideration of Bulk Components of the Diet

Water

Details of approximate water requirements are given elsewhere (Table VI) and it should be stressed that an insufficiency of water may lead to clinical upsets which might not have appeared if sufficient water had been available to ensure that the bird was able to excrete excesses of intake—*e.g.*, salt poisoning. It is unlikely that an excess of water intake would lead to disease conditions.

Water is particularly important in young chicks and turkey poults which may have to be encouraged to drink by using coloured containers (green has been used with success). Sudden drops in egg production can be caused by a gross restriction of water intake.

Energy

It is unlikely that, under normal feeding conditions, any deficiency of energy would arise. However, starvation can, and does, occur occasionally when sufficient trough space is not provided. The initial signs of starvation are obvious but, in the later stages, the bird may become so weakened that death will be due to crushing.

The fatty liver syndrome in battery housed birds has been associated with high energy diets, as have a number of mineral and vitamin deficiencies.

Protein

There is little evidence to suggest that excess of protein will cause specific disease but visceral gout may be an associated condition. Occasionally nephritis has been related to excessive protein intake, but the evidence is not extensive.

Deficiency of protein will lead to reduced growth rate but this is not a problem likely to be encountered by the practising veterinary surgeon. It has been shown on numerous occasions that adult laying stock can lay well on diets with 14 per cent. of protein, or less, but this will only be true if the energy content of the food is not excessively high.

Lysine deficiency in turkeys is, however, a specific condition resulting in poor growth. The condition can be recognised by the lack of pigmentation in the wing feathers and at least 1.2 per cent. of lysine is necessary for normal growth (Fritz *et al.*, 1946).

Fibre

An excess of fibre can cause illness among poultry and the main circumstances under which this is likely to occur are:—

(a) Chicks or growing poultry on range may not have an adequate supply of concentrates or, alternatively, management may not allow birds sufficient opportunity to consume adequate amounts of an apparently ample supply—*e.g.*, lack of trough space. Under either circumstance the poultry must look elsewhere for nourishment and long fibrous grass may be eaten in excessive amounts.

(b) Birds housed intensively on straw, wood shavings or similar material may consume large quantities of this litter if conditions are much below standard—*e.g.*, overcrowding.

(c) In an attempt to delay sexual maturity of pullets high fibre rations are now being fed on a number of farms. The fibre content of these rations is approxi-

mately 15 per cent. and should they not contain sufficient minerals and vitamins, deficiencies will occur if the ration is not consumed in adequate amounts.

Abnormalities of the Digestive System

Thirst

In the majority of cases excessive thirst will be associated with an insufficient allowance of water, lack of water trough space, or as a reaction to high intakes of toxic substances which are eliminated by the kidneys, *e.g.*, salt.

Anorexia

As far as nutritional disease is concerned, anorexia is usually one of the terminal signs of a gross deficiency of a specific nutrient, *e.g.*, a vitamin.

Depraved Appetite

Most cases are due to mineral imbalances in the diet, especially calcium or phosphorus, but depraved appetite can be associated with nervous disorders, such as nutritional encephalomalacia, when foreign material can be found in the crop and gizzard. Occasionally, depraved appetites are due to lack of trough space and mild intestinal disorders.

Pendulous Crop

This is mainly due to an excessive secretion of the mucous glands of the crop. It can be ascribed to some form of enteric upset and sometimes to an excessive intake of calcium. A number of cases have been associated with fungal growths in the crop resulting from the ingestion of stale or fermenting food.

Impacted Crop

Often a sequel to pendulous crop and other non-specific alimentary disorders causing a depraved appetite. Some crop impactions have been associated with fowl paralysis and even nephritis. However, the condition is probably more commonly due to the excessive intake of fibrous grass when birds are on range. The risk can be reduced if the sward is kept short and sufficient alternative food and trough space are provided.

Treatment of pendulous or impacted crops can be attempted by manipulation of the crop with the bird held head down. Should this fail, it is a simple matter to empty the crop by direct surgical methods. Small quantities of food and water should then be allowed at frequent intervals, and if the condition recurs, destruction of the bird is probably indicated.

Impaction of the Intestine

This condition is generally diagnosed only at *post-mortem*, but it may be present in flocks where cases of impacted crops are seen. If it is decided to treat the flock then Epsom salts are usually prescribed in doses of about 1 lb., per 150 lb., live-weight of birds.

Wet Droppings

Sudden changes in the diet can cause increased water content of the excreta, possibly as a result of a mild enteritis. Excessive salt intakes will almost certainly result in diarrhoea of varying severity. Contrary to general belief, there is no indication that properly balanced high energy low fibre rations are associated with diarrhoea. Consumption of high levels of insoluble grit may lead to a large number of small pieces being carried by the intestine and this may give rise to diarrhoea due to the resultant irritation.

Gizzard Erosion

Degenerative changes in the muscles of the gizzard may be noted as greyish patches—a condition related to vitamin E deficiency. A more advanced form occurring as dark coloured rough depressions has been related to a deficiency of vitamin K (Stafseth, 1958).

Fatty Liver

This condition is associated with excessive energy intake and is probably more common in present times as a result of high energy laying foods and intensive housing conditions. There is some evidence that a few cases may be due to a riboflavin deficiency while others have been related to deficiencies of vitamins E, B_{12} and methionine (Trouw, 1959).

Usually only individual birds in a flock are affected, although a substantial number of cases may occasionally occur in battery housed birds either

(a) where an *ad lib* supply of food is allowed, or

(b) in multiple cages where the "boss" may consume more than its share.

Affected birds suddenly go off lay despite their healthy appearance. Such birds may seem heavy when handled and the enlarged liver may be detected on palpation.

Treatment should be directed towards an immediate curtailment of the ration, and an enquiry made into the feeding and management of the unit. One should not necessarily change to an inferior food as this may result in an over-all drop in egg production which can be of greater economic importance than the loss of a few birds.

Abnormalities of the Head

Vitamin A Deficiency (Nutritional roup)

Lesions of the head of nutritional origin are, in the majority of cases, related to a deficiency of vitamin A. The eyes show excessive lachrymation, cheesy accumulations and occasionally xerophthalmia. The nostrils show similar watery exudates which gradually become cheesy in nature. Small pustules and ulcers covered by a thick exudate can be seen inside the mouth. Pale kidneys and uric acid deposits may be observed on *post-mortem* examination.

Nutritional roup of the adult is typified by the above symptoms with the addition of respiratory symptoms associated with exudates in the upper respiratory tract.

In all cases of vitamin A deficiency the birds become lethargic and weak followed by incoordination of the limbs similar to the ataxia seen in birds affected by crazy chick disease (Scott, 1957). A general ruffled appearance will be presented and hatchability of eggs will usually be detrimentally

affected. It is important to remember that birds deficient in vitamin A may be more liable to many other specific conditions due to their reduced ability to resist infections.

Treatment by providing a suitable source of vitamin A will usually be effective.

Chick Dermatitis

Mention is made of the association of pantothenic acid deficiency with chick dermatitis, but this condition does not seem to be very common. Scaliness occurs at the corners of the eyes and mouth of affected chicks as well as around the vent. Dermatitis also can be seen on the feet of chicks but not in turkey poults.

Mandibular Disease

This disease is generally associated with dry mash feeding especially if the food has been finely ground. Pasting of the food on the mandibles results in ulceration, necrosis and an accumulation of necrotic material. Sometimes localised necrosis of the tongue can be seen.

Wet mash or whole grain feeding will generally effect a cure, especially if the removal of large accumulations of necrotic material are removed.

Abnormalities of the Skeleton

Rickets

As is well known, this condition is due to an imbalance of calcium and phosphorus and/or a deficiency of vitamin D in the diet. It should be noted that calciferal (vitamin D_2) is not utilised efficiently by the fowl and it is therefore essential to supply vitamin D_3.

A detailed description of the condition is not included in this paper as it is assumed that the clinician is already aware of the most important features, *viz.*, ruffled appearance, leg weakness, painful swollen joints followed by softening and distortion of the skeleton especially the legs, sternum ribs, and a rubbery condition of the beak.

Osteomalacia

As a result of lack of vitamin D or an imbalance of calcium and phosphorus intakes, the production of soft-shelled eggs may be the first sign of the disease. Fragile bones and soft beaks will follow. In any investigation of this disease one should ensure that there is a suitable total intake of calcium in the diet of laying stock.

Treatment of rickets and osteomalacia should be directed at correcting the levels of calcium, phosphorus and vitamin D_3 in the food.

Perosis and Hock Disorders

As time passes the aetiology of these conditions seems to become more involved. Various factors have been associated with each other in the production of perosis (slipped tendon) such as an imbalance of calcium and phosphorus, deficiencies of manganese or choline and more recently a deficiency of zinc. When excessive amounts of calcium are present in the diet it seems likely that the chicks' requirement for manganese and zinc is increased. Housing may also play a part as it has been shown in one experiment (Cummings & Tribe, 1956) that birds reared on wire developed 60 per cent. more cases than those reared on wooden floors. However, the diet in this case was considered to be deficient in choline.

Affected chicks initially show small haemorrhages on the hock joint which then becomes enlarged. The metatarsus rotates and eventually the tendons slip off the joints often resulting in complete inability of the birds to bear any weight on the affected leg

In turkeys, leg weakness as distinct from perosis has been described (Comben, 1959; Blount, 1960) occurring at about the 4th and 16th weeks of life. This has been ascribed to deficiencies of numerous vitamins but especially to vitamin E, choline and nicotinic acid. Various minerals have also been incriminated.

It would seem that no single factor can be defined as the cause of perosis and other hock disorders and therefore each case should be judged according to its individual circumstances.

Curled Toe Paralysis

This condition is caused by a deficiency of riboflavin which is the most important member of the B complex in poultry rations. Deficiencies in adults are most often reflected in the hatchability of eggs. (See section IV.)

Chickens reared on diets with suboptimal levels of riboflavin will usually begin to show signs of leg weakness at about 2 to 3 weeks of age. This is followed by the curling of the toes into a clenched position and often the birds will sit on their hocks. Poor growth rate and ruffled feathers are features also. Turkey poults develop a dermatitis round the beak and on the foot pads.

Usually the incidence of curled toe paralysis is greatest when birds are reared on wire floors due probably to a greater stress on the legs and also to the fact that riboflavin can be synthesised by bacterial action in the excreta of the fowl.

Affected birds will generally show a spontaneous recovery due no doubt to a decreasing requirement with age, and also as a result of coprophagy. The administration of yeast preparations to affected birds will aid quick recovery.

Gout

See visceral gout (systemic abnormalities).

Cage Fatigue

This condition develops among battery birds in full egg production which become unable to stand upright and often sit down on their hocks. It is due to a deficiency of dietary calcium—usually where the total intake is less than 2.5 per cent. of the ration (Fisher, 1956). Most birds will recover if put on the floor and adequately fed.

Nervous Abnormalities

Nutritional Encephalomalacia

Crazy chick disease, as the condition is commonly called, is due basically to a deficiency of vitamin E. Affected chicks may show signs from 10 days to

about 6 weeks of age. Some birds have been known to recover spontaneously but death usually takes place in 12 to 24 hours. Intermediate symptoms are wing flapping, ataxia, loss of equilibrium, and paddling movements of the legs when birds are lying on their sides. A characteristic push over backwards is seen when support is removed from an affected bird in a standing position (*Handbook on Poultry Diseases*, 1948).

The aetiology of crazy chick disease is extremely complicated and a number of unconnected factors are involved. Male birds seem to be more susceptible than females (Singsen *et al.*, 1954), some breeds more than others (Carneghan & Markson, 1956), and birds reared on wire have not shown signs, although others from the same hatch have developed the disease when reared on litter. Thus "physiological stress" may be responsible for increasing the chicks' requirements for vitamin E, even although the chicks' theoretical requirements of 9 to 25 mg. per lb. of food (Titus, 1951) are being met in the food.

Vitamin E is an antioxidant and it can be partially replaced in the diet by other antioxidants. It seems, therefore, that the function of vitamin E is as an antioxidant either by:

(a) reducing oxidation of fat in the food, resulting in destruction of fat soluble vitamins;

(b) being absorbed and utilised by the body as an antioxidant, especially in the liver.

At the risk of over-simplification, an attempt has been made to list the 3 main factors involved in the production of crazy chick disease, working either individually or in unison—

(1) Vitamin E deficiency of hatching eggs.
(2) Vitamin E deficiency of chick rations.
(3) Physiological stress—*e.g.*,
  (a) Management and environment—wire or floor rearing.
  (b) Type of food—pellets, crumbs or mash deficiency of other vitamins. High or low energy.
  (c) Breed of chick—heavy or light.
  (d) Sex.
  (e) Rate of growth.
  (f) Presence of other disease.

Undoubtedly crazy chick disease is due to a deficiency of vitamin E as the condition can be prevented or cured by the addition of small quantities of the vitamin to the drinking water—(24 mg. vitamin E in 1 gallon of drinking water has been used as the daily treatment for 100 birds of 3 weeks of age). However, it is most important to investigate the other factors discussed in order to prevent further outbreaks.

Gross Systemic Abnormalities

Visceral Gout

Dietetic upsets and errors in management are probably the main predisposing causes of this condition which may be diagnosed *post mortem* by the deposition of urates on the serous surfaces of the abdomen. Chronic cases may show these deposits in the joints of the legs.

The main reason why an excessive protein intake is suggested as the primary cause of visceral gout in the fowl is that excess nitrogen products of metabolism are excreted as uric acid. Any factor causing excessive uric acid production or renal inefficiency is, therefore, likely to lead to the deposition of unexcreted uric acid in the tissues as urate.

Excess of protein is unlikely to be the sole cause of visceral gout as this theory is not supported by experimental evidence. However, prolonged high energy intake or prolonged feeding of foods in which the oil content contains a high proportion of free fatty acid might lead to fatty degeneration of the kidney cells resulting in inefficiency.

Haemorrhagic Disease

One cause of this disease has been reported as a deficiency of vitamin K resulting in a slow clotting rate. The condition has also been associated with the use of sulpha derivatives as coccidiostats.

Haemorrhages of varying degree may appear anywhere in the body—*e.g.*, under the wings and on the combs. On *post-mortem* examination there are multiple haemorrhages in the subcutaneous tissues, particularly on the breast and thighs, on the wall of the intestine and in the heart and spleen (Gordon, 1959).

Exudative Diathesis

Exudative diathesis in a condition of poultry apparently caused by a deficiency of vitamin E and is more common in turkeys than chickens. The skin becomes reddened, especially under the wings, followed by subcutaneous oedema. The oedematous exudate may also be seen in the pericardium.

It is thought that, as a result of a vitamin E deficiency, there is an increased permeability of capillaries thus allowing diffuse haemorrhage and exudation of plasma (Dam & Glavind, 1942). There is some evidence that the condition can be controlled by the feeding of dried brewers' yeast in maternal rations or alpha tocopherol (Creech *et al.*, 1957 A and B), and one wonders whether this might indicate that a deficiency of members of the "B" group of vitamins can play a part in the aetiology of diseases related to vitamin E deficiency on diets which apparently contain enough of this vitamin.

Section III: Abnormalities of Egg Shell Quality

Poor egg-shell quality can be related in most instances to some error in nutrition, although it must be remembered that laying stock affected by any disease may produce eggs with an abnormal shell texture. For the production of first quality egg shells, the laying bird must be supplied with adequate amounts of calcium, phosphorus, magnesium, manganese and vitamin D. It should be noted here that there is unlikely to be a deficiency of magnesium in normal diets.

Thin-shelled eggs are usually the result of a deficiency of calcium or vitamin D, while misshapen eggs with or without wart-like growths are related more to an imbalance between the essential requirements. It is most important that calcium supplies are adequate from at least 2 weeks before point of lay.

Section IV: Abnormalities of Hatching Eggs

The nutrition of adult breeding stock is worthy of special mention as small increases in hatchability of eggs can lead to substantial increases in profits—*e.g.*, by raising the hatchability by 1 per cent. in eggs produced by 2,000 hens over a period of 4 months, there can be an extra 1,500 chicks hatched if settable egg production is approximately 60 per cent.

Generally speaking, breeding stock nutritional requirements are similar to those of other adult laying stock with slightly higher margins of safety for vitamins. However, it is felt that special mention must be made of a small number of points.

Vitamin E

Rations deficient in vitamin E can reduce fertility and cause embryonic death at 4-5 days (Coates, 1949) due to perivascular haemorrhage. Deficiency of vitamin E in breeders' diets has already been seen to have some part in the actiology of crazy chick disease. The level of vitamin E in turkey breeders' diets is even more critical than in fowls' diets (Funk & Irwin, 1955) as shown by the following :—

53.7 per cent. hatchability with control deficient diet.

81.9 per cent. hatchability with control diet + vitamin E (no levels given).

Riboflavin

Deficiencies of riboflavin in breeders' foods can result in the typical "clubbed down" condition seen in embryos in the later stages of incubation. Embryonic death may take place at approximately 4 and 14 days.

Other Vitamins

Pantothenic acid, choline, nicotinic acid and vitamin B_{12} are also important in obtaining good hatchability, but in normal practical rations they are unlikely to be deficient.

Calcium

Evidence suggests that excessive amounts of calcium in the diet will decrease hatchability. It would seem, therefore, that it is best to limit the total calcium allowed to breeding stock to about 3.25 per cent. of the food intake.

Manganese

A deficiency of manganese will cause heavy embryonic mortality at about 20 days. The embryos will show micromelia (short legs and wings), domed cranium and parrot beaks. A deficiency of biotin can also cause parrot beaks and short bones, but webbing of the toes and slipped tendons are usually also present.

Section V: Relationship Between Nutrition and Disease

It is important to bear in mind that there is no clear-cut dividing line between an animal in a normal state of health, if this can be defined, and that animal in a state of disease. There is a gradual shading from one condition to the other and the subclinical stage may not be recognised. This observation is certainly true of nutritional diseases, and it would seem that here we have the starting point of a line of investigation of practical value in the understanding of the important relationship between nutrition and disease.

Much work has already been done on this subject and a number of relationships have already been postulated—a few of which are quoted :

(1) Between ascarids and vitamin A—a deficiency of vitamin A favours an infestation by *A. galli* which may then damage the bowel, thus reducing its ability efficiently to absorb the vitamin A activity contained in the bowel (Pande & Krishnamurty, 1959).

(2) Between coccidiosis and vitamin A—low levels of vitamin A may increase the likelihood of coccidiosis of poultry (Erasmus *et al.*, 1959).

(3) Between coccidiosis and vitamin K—the addition of vitamin K to poultry diets reduces the number of deaths from coccidiosis (Otto *et al.*, 1958).

(4) Between diet and *p*H of bowel contents—fat or whey in poultry diets are likely to lower the *p*H of the bowel contents, at least in certain sections (March *et al.*, 1958).

(5) Between incidence of blackhead and *p*H of intestine—if the content of the gizzard is acid, the incidence of blackhead will be reduced, while it is increased if contents are alkaline (Horton-Smith & Long, 1956).

A number of other possible relationships receive mention in a review article (Chubb, 1958) and it would seem that while advances can be expected in our knowledge of nutrition and of nutritional disease, much greater importance might possibly be put on to the relationship between nutrition and disease along either of the following two lines.

(a) The effect of nutritional deficiencies and the resistance to disease.

(b) The effect of variation in the condition of bowel contents, induced by changes in feeding, on the bacterial and parasitic flora.

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THE SPEAKERS' INTRODUCTION

The Chairman, Professor J. S. S. Inglis, said that it was somewhat presumptuous for him to occupy the Chair on an occasion like this as he had worked to only a limited extent in the poultry field. However, he accepted the position only after being assured that certain members of the profession who were distinguished in the field had been invited to do so but had not found it convenient on that particular day.

The size of the poultry industry should be emphasised. A few years ago poultry was regarded as the perquisite of the womenfolk on the farm. To-day, after a few years of rapid expansion, it had emerged as a very substantial part of the agricultural economy. The present rapid expansion was very well seen in the broiler industry which had an annual output of 75 million. It was expected to be 250 million by 1965.

Without the veterinary profession that tremendous expansion could not have taken place. It was due to the fact that veterinary workers had been able to bring certain diseases under control. The importance attached to poultry disease was illustrated by the recent appointment of a committee by the Ministry of Agriculture and the Secretary of State for Scotland to inquire into the control of fowl pest, and the meeting would no doubt wish to offer congratulations to Professor Robertson of Edinburgh on being appointed the veterinary representative on that committee.

There were to be contributions by 4 well-known experts in the poultry world and they would present papers on a wide variety of poultry disease and nutritional problems.

Professor Inglis then called upon Mr. J. D. Blaxland to present his paper.

Mr. Blaxland said that it was always a pleasure for a specialist to talk on his own subject but in addition he felt that it was a particular honour to give a paper that morning. He might have wished, however, that the subject was one which could be more easily presented. The paper was difficult to write, not because one did not know what to say but because there was such a tremendous mass of material to draw upon and put forward.

The basis of the paper was a short account of some of the more important infectious diseases excluding those covered by the others taking part in the symposium. He had in mind the needs of those in private practice, who felt that they would like, or that they ought, to take more interest in the poultry problems of their clients. It was a mistake for a veterinary surgeon to go out into the poultry field thinking that, with a little knowledge of the infectious diseases, he would be able to see his way through the multiplicity of problems that he would meet. It was not as easy as all that.

The organisers of the programme did well to align a symposium which emphasised that infectious diseases were not the only cause of economic loss. The relative importance of the parts played by a disease agent and the environment of the host was always open to speculation, but the training and experience of the veterinarian should enable him to assess more easily the significance of the various clues which might provide the key to the problem. Even so, it was not always possible to diagnose a specific disease and initiate control measures for it.

Disease was not simply a matter of specific infections. The question, in most outbreaks, was rather: What mistakes and errors have been committed on this farm to produce a disease problem amongst the birds and what are the factors which account for their inability to withstand the normal stresses and strains of living there? That approach was more likely to be profitable than was a searching of mind and notes for the description of a specific disease to fit the picture presented.

He would like to think that some, at least, of the success that had been achieved in controlling poultry disease was the result of work done by his predecessors in the poultry field. But it was also true to say that the modern poultry industry could not flourish without the achievements of research workers and the skill of manufacturing chemists in producing a steady flow of antibiotics and drugs which checked a number of diseases which would undoubtedly occur on a serious scale if those remedies had not been available.

On the other hand, the almost universal use of drugs on poultry farms to-day brought complications which had not been foreseen. On some occasions it tended to suppress disease or to distort the picture and increase the difficulty of diagnosis. Furthermore, there was the development of resistant strains of certain organisms to antibiotics—so far without evidence that that resistance was tied to increased pathogenicity. When looking into a poultry problem the veterinarian should not overlook factors such as genetical make-up and environment of the flock. There was a good deal of evidence that genetical resistance occurred in certain strains.

Deficiency diseases were not the subject of this paper, but time and again one was faced with what would appear to be a low plane of nutrition, not sufficient to bring about symptoms of deficiency disease but which seemed to lower the resistance and to prevent birds from withstanding the ordinary conditions of their environment and perhaps to permit more rapid build-up of infection leading to an outbreak with serious economic results.

The environment and siting and design of houses were of importance. In some of the more specialised branches of the industry, the importance of scientifically designed buildings was realised and that side of the business was in the hands of specialists who knew a great deal about such things. But there was a great deal of makeshift poultry housing to-day, particularly when profits were attractive to people who thought that by minor alterations to existing buildings they could turn themselves into modern broiler producers or keep a profitable flock of laying birds.

It was essential that the veterinary surgeon who wanted to extend his activities into the poultry field must acquire that amount of knowledge of poultry rearing and management which he would consider necessary before he ventured to express an opinion on mortality and unthriftiness in the other animals of his clients. More than 60 per cent. of the mortality among baby chicks and turkeys was not due to specific disease but to factors which were concerned with some aspect of management, starting in the hatchery, and conditions during transit from the hatchery, to conditions in the first few days on a customer's premises. Without some knowledge and appreciation of the difficulties that had to be met and overcome and of the elementary hygienic measures that must be adhered to, the veterinary surgeon was going to be very much at a loss when advising his clients about mortality.

That did not mean that laboratory examination was not necessary and it was essential in the diagnosis of the specific diseases, but a careful survey of environmental conditions and circumstances under which losses were occurring would often enable the experienced observer to give his clients useful and helpful advice. Bacteriological examinations were essential in early chick mortality, since, quite apart from the risk of a mistaken diagnosis, laboratory confirmation was required by most hatcheries in support of claims from their customers for compensation.

Mr. Blaxland said that it was impossible to get accurate figures of the loss caused by various diseases or even by the whole range of diseases. Figures of interest, from his own *post-mortem* room were: In 1959, only 14 per cent. of the adult birds examined were infected with specific disease, excluding leucosis, and approximately 20 per cent. of the batches of chicks. Last year, Gordon had tried to assess the monetary loss from diseases in respect of the broiler industry and had quoted £5 to £15 on a crop of broilers. Reference was made to estimates of the British Broilers Growers' Association, which indicated that a broiler producer with 8 per cent. mortality in the last week of a crop of broilers would only just cover costs of production, and that if the rate were 10 per cent. or 12 per cent. he would lose money. Quite recently in a report in THE VETERINARY RECORD Gordon estimated that the disease loss annually was in the region of £20 million as against an annual value of about £200 million.

The Chairman then asked Mr. Gordon to present his paper, saying that they were always especially happy to welcome their colleagues from Northern Ireland.

Mr. Gordon expressed sincere appreciation to the Organising Committee for inviting him to present a paper, and congratulated them on their foresight in focusing attention on this all-important field of disease. The paper briefly reviewed the economic importance of the poultry industry in relation to agriculture as a whole and the magnitude of the losses occasioned by leucosis and fowl paralysis. An endeavour had also been made to give the more salient points regarding the *post-mortem* findings in the diagnosis of these diseases.

At Stormont, of the 2,410 diagnoses made as a result of examining 5,379 adult specimens during 1959, approximately 23 per cent. were of neural fowl paralysis, while 60 per cent. of diagnoses in this age group were accounted for by leucosis, fowl paralysis and tumours. Those figures might be slightly higher than those for Great Britain as a whole but they were reasonably representative. The practitioner, therefore, need have no qualms about the part he could play in diagnosing these diseases. It was hoped as a result of the points set out in the paper, which really formed a digest of other workers' findings, that the practitioners would be in a better position to help in this field.

Mr. Gordon then showed some transparencies.

The Chairman said he did not think Dr. Horton-Smith needed any introduction. All through his veterinary career the name of Horton-Smith had been synonymous with coccidiosis.

Dr. Horton-Smith said that he was glad to be taking part in the discussion. The paper as submitted skipped very lightly over the biological features of *Coccidia* as a whole. He now wanted to emphasise the points of diagnosis of the pathogenic species of *Coccidia* and the methods of control. A lot of the reports that were made about medicaments came from America, and only the previous year, while there, he saw that American strains did not always respond to drugs in the same way as others did.

There were, roughly, 4 coccidial diseases of poultry caused by different species, and one might refer to them by the collective term coccidioses because of the differences that occurred in the epidemiology and virulence of the associated species. Two species had now become important as causes of disease although a few years ago they were regarded as relatively harmless. Other species, now relatively harmless, might eventually become extremely important as causes of disease in foods maintained intensively.

A series of slides illustrating severe parasitism by some species was then shown. The merits of the different anticoccidial agents were discussed in relation to the species described.

Dr. Horton-Smith went on to say that work was going on at several centres on the difficult problem of immunity. A very interesting stage had been reached at Houghton where antibody to *Eimeria tenella* have been demonstrated in the serum. The presence of serum antibody is probably an indication of resistance and may not play an active part in protecting chickens from infection.

In America oocysts were being fed to chickens with a view to producing resistance. A superior quantity of different species were sent to the farmer who poured them into the ration. It was likely that the stronger birds got more than their ration; these birds passed a new generation of oocysts on to the litter which the chickens picked up. Treatment was then administered which protected the birds from disease and allowed immunity to develop. Oocysts of all species were being brought on to a farm and it seemed to be a "hit and miss" business. It was advisable to proceed with caution before the method is adopted in this country.

The Chairman then asked Mr. W. W. C. Gibson to introduce his paper.

Mr. Gibson said that he was conscious of the honour of being asked to present a paper, and of the fact that there were probably a number of other people who would be much better qualified to do the job. First, it was only right to endorse what Mr. Blaxland had said about the interrelationship of management and nutrition, housing and disease. A poultry problem would usually relate to more than one factor and all factors must be considered together.

The slides presented had mostly been chosen to illustrate the main features. The first 3 gave an indication of the economic importance of the poultry industry as a whole.

Slide: Tables I and II showed the growth of the poultry industry over the last 10 years and demonstrated that the number of table chickens produced annually had increased at a rate that was quite fantastic.

Slide: Table III illustrated that during 1957 and 1958 the compensation paid for fowl pest slaughter was very much more than that paid in respect of either foot-and-mouth disease or bovine tuberculosis.

Slide: Table IV showed that the tonnage of compounded feeding-stuffs for poultry was now greater than that for cattle or pigs.

Slide: Table V. Not only had the industry grown but the expected egg production per bird had risen from 145 to 180 in 7 or 8 years.

Slide: Table IX served as an example of the various tables of nutritional requirements shown in the paper. As the exact requirements were not known for all breeds under different environments, these tables must be regarded only as guides.

Slide: Table XII. Figures from the Norwich Investigation Centre showed that nutritional diseases did not represent a high proportion of poultry disease. Of specific conditions of nutritional origin encephalomalacia was the most common.

Slide: In considering nutritional encephalomalacia, one must look for factors in management which might have caused a deterioration in the food—was the food too close to the light, was it getting wet, and so on? The various factors associated with physiological stress must also be borne in mind. Basically encephalomalacia was due to a deficiency of vitamin E, but one must discover why this deficiency has occurred.

Slide: Where disease was suspected to be associated with the food, analysis of food might be necessary but the limitations of analysis should be realised. Unless one expected to find a specific condition it was extremely difficult to decide whether or not the food was likely to be involved in the outbreak of disease. If poisoning was suspected the laboratory should be told the group of poisons suspected. In a general analysis of food, oil protein and fibre would seldom give an indication of whether food was at the root of the trouble. If the salt content was greater than about

.75 per cent. one might suspect its being too high. If the free fatty acid content was higher than 40 per cent. the food might be deteriorating. If the peroxide value was higher than 10 one might assume that the food was rancid. However, these values would be constantly changing and such changes were often in the opposite direction.

Slide: Showed poultry on deep litter; 6,000 potential laying birds in a house, in the U.S.A., about 50 yards long. There was automatic feeding around the house, as well as automatic water. Nesting boxes faced alternate ways throughout the length of the house. When the birds laid eggs, they rolled on to a conveyor belt, and when an operator pressed a button the eggs came towards him for collection. There was presumably a great disease risk in the house and, if the attendant was not energetic, he might not see one end of the house from one week's end to the next. This is the type of problem which could well confront us in this country in the near future.

The industry to-day required a middleman to relate the findings of the laboratory to the particular aspects on any farm on which disease occurred. The laboratory could not interpret their findings in respect of an individual farm; nor could the farmer. Somebody should do it. It should not, however, be a representative of one of the commercial houses, whether a sales or technical representative. The veterinary surgeon in practice must recognise the importance of the poultry industry and take his rightful place within it. In effect, he must become the middleman.

The General Discussion

Mr. K. W. Aspinall (Nyasaland) said he was greatly interested in the papers. In Great Britain it did not seem to be fully realised how far the economics of the poultry industry had affected treatment. Only a highly developed industry could afford antibiotics. A lot of birds were sent out to African owners and the treatments must be done before they were sent out because they could not afford any antibiotics. The birds must be healthy and able to survive their environment once they had been despatched.

Poultry diseases spread through the country very rapidly. Newcastle disease was experienced in 1955 for the first time in some places there, and within 2 years it was all over the country. Coccidiosis had been rare but that too was now all over the country. The American business of putting out oocysts was noteworthy. Drugs were not the answer and it was difficult to know what to do. Perhaps the authors could say something about the latest information on the prevalent species in Africa. The strains in America were different from those of the U.K. species. Might not a similar difference apply to Africa?

Dr. Horton-Smith said he had some experience of actual strains, *E. acervulina* and *E. maxima* from Africa and their reactions to certain forms of medication had differed from those experienced in the United Kingdom but sulphonamides gave good control to both species. The usual medicaments might not control some diseases under African conditions. One could be fairly sure that there were no species in Africa that did not occur in, say, America but, of course, there might well be strain differences which might account for the difficulties mentioned by Mr. Aspinall.

Mr. M. H. Fussell (Cambridge) said he was pleased that it had been decided to have 4 papers on subjects requiring especial attention. What did Dr. Horton-Smith consider was the most favourable form of prophylactic treatment and therapeutic treatment for *E. acervulina* and *E. maxima*? And would Mr. Blaxland say what was the best treatment for pasteurellosis in turkeys?

Dr. Horton-Smith said that, so far as *E. acervulina* and *E. maxima* were concerned, sulphonamides were the best drugs in both cases. Unfortunately, those drugs had side effects if given over long periods. Methiotriazamine was said to control these species and 1-(4-amino-2-n-prophyl-5-pyrimidinylmethyl)-2-picolinium chloride hydrochloride certainly controlled *E. acervulina* when used prophylactically. At least one strain of *E. acervulina* and several strains of *E. necatrix* were effectively controlled by 3, 5-dinitro-*o*-toluamide.

Mr. Blaxland said that he had referred to treatment in his paper.

Mr. Fussell said that he was really interested in prevention.

Mr. Blaxland said that *Pasteurella* vaccines were used to some extent in other countries but he had no experience of their use in Great Britain. Perhaps some of the visitors from abroad could enlighten them. He had *Pasteurella* outbreaks very much in mind when making his general remarks because *Past. aviseptica* was quite frequently isolated from individual birds. Treatment of the individual bird by injection with antibiotics was useless when that individual was already infected. There was much difficulty in picking out ailing birds from a flock of thousands, and when one came to treat them, more often than not it was too late. A practitioner called in to advise on treatment might feel bound to make an attempt to do so, but it was better not to be too sanguine about the results.

Mr. G. V. Short (Wilmslow) said that Dr. Horton-Smith had referred to the difficulties which arose from the different susceptibilities of coccidial strains in America and elsewhere, and Mr. Aspinall from Nyasaland had emphasised these. A great deal of bacteriological effort had been put into the establishment of the National Collection of Type Cultures in the U.K., and the American Type Culture Collection; but there was little, if any, concerted effort by the workers concerned with coccidiosis to establish and maintain a similar type collection of coccidial strains on which could be based screening tests for present or future drugs. Could Dr. Horton-Smith say what was intended, if anything, to set up a coccidial type collection?

Dr. Horton-Smith said that an American group was doing something in this connexion. The question was discussed with him last year and many difficulties were involved. It was known that strain differences existed between species. In compiling a library one had to determine not only the species but the strain. Any published work on a strain would relate to that one strain. A tremendous amount of labour was needed to compile such a library. At present the bird must be used as the test tube and it must be kept entirely parasite free before infection and protected from extraneous infections during the period of oocyst collection. There were many gaps in the organisation at the moment, but such a centre might certainly be of value. He did not know how far the American group had got but he would let Colonel Short know later.

Mr. N. Comben (Surbiton) asked whether Mr. Gibson could give guidance as to the different F.F.A. and peroxide values which would be acceptable in feeds for adult birds, layers and breeders, as opposed to those acceptable in the more critical types of feeds used for young stock and for rapidly growing birds like broilers and young turkeys.

Also, what was his opinion of the recent suggestion that the use of the grain sorghum milo might be partly responsible for the mysterious outbreaks of disease currently affecting turkeys all over the country?

Mr. W. W. C. Gibson thought it obvious that adult stock would be affected less by alterations in the condition of the oil content. It was not really possible to lay down safety margins unless one went right down to something like 35 or 30 for FFA and to a peroxide value of perhaps 5. However, for adult stock a FFA of 50 should be acceptable and for younger stock it was desirable to go under 40.

Mr. Comben had no doubt been referring to a recent article on milo. It was unlikely to be proved an offending article. It was likely to be dangerous only from the point of view of the possible production of prussic acid in grain grown under extremely dry conditions. 1959 was not a particularly arid year and most of the American milo is grown under irrigation. In any case, it was not likely to consist of more than 25 per cent. of any ration.

Dr. Horton-Smith said that there were 2 species in this country which were pathogenic to turkeys. He had not dealt with the problems of prophylactic medication and

correctives in turkeys to any great extent, but had rather worked on the lines—perhaps wrongly—of not medicating until the disease had been definitely diagnosed in the birds, and then 2 sulphonamides had been applied with great effect.

Dr. A. W. Stableforth (Weybridge) said that the time was very opportune for veterinary surgeons in practice to take a greater part in the control of poultry disease. The point had been very well made that the majority of it was not due to specific bacterial and virus causes but to husbandry and a variety of factors about some of which not much was known.

Veterinary surgeons in practice should play exactly the same part in regard to poultry as they played in regard to cattle, sheep and pigs. They could go on to the farm, consider all the conditions on the farm, as they were able to do in a way no one else could, and act as a sieve or screen for the laboratories. There was going to be room for practitioners to be called in more than in the past.

That brought up the point of poultry training. Inquiries had recently been made about what could be done because the training at college was perhaps not adequate or could be increased, and people were asking for facilities for refresher courses. What other means could be used to help those in practice to equip themselves with more knowledge of husbandry as well as of disease, so that they might really take their proper part in this field?

Dr. Horton-Smith felt that he could speak in a disinterested way. Some laboratories were having visits from practitioners who were specialising in poultry matters within groups. Personally, he had enjoyed having them very much because of their keenness and the rapidity with which they absorbed the information in question. There was the question of the post-graduate training that had come into force at Wye. It was a full-time course which made it quite impracticable for the general practitioner. It enabled people, after taking their veterinary or other qualification, to proceed to take a degree in poultry science at the University of London.

Mr. Blaxland said that quite a lot of thought had been given to the problem of how best to help the practitioner to play a bigger part in poultry. Individual veterinary surgeons in practice had had short spells at Weybridge, but that was not by any means the solution to the problem. The number must be limited because the organisation was first and foremost that of a routine diagnostic laboratory and it was not possible to do the work and instruct and lecture to a group of people at the same time. Whether it would be possible to organise a series of short courses based on universities using the centres as a source of material, remained to be seen. It was unlikely that practitioners would want to send their assistants for more than a few days on that sort of course.

However, the practitioner could go a long way during the course of his everyday work towards filling this gap in his education. One could pick up a great deal about the management of an unfamiliar species merely by showing an interest in the subject when on a farm for other purposes. Surely it was not detrimental to the practitioner's status to say, "I see you have a new broiler house or deep litter house; that's interesting—I was reading about it the other day. Will you spare 10 minutes to explain the system to me?" In that way quite a lot of information on poultry management could be acquired. After a comparatively short time, they would begin to feel much more confident when faced with poultry problems. The remedy was largely in their own hands.

Certainly more should be done by the veterinary schools in organising poultry lectures and field visits, etc., in order to provide a course which had so far been missing and to awaken the interest of the potential graduate in this important branch of agriculture. Much of the remedy however was in the hands of the individual who could acquire knowledge for himself by keeping his eyes open and asking questions.

Dr. Stableforth said it had not been intimated that nothing could be done at Weybridge or at V.I. centres. At this stage all concerned needed to know what other people wanted? It might be possible in many practices for a principal or assistant to go away for a week or fortnight to study poultry diseases because it was so important; in other practices the view might be taken that courses spread over shorter periods were the best answer.

The Chairman said that the greatest value had been obtained by his colleagues from co-operating with the local agricultural college. In the South-west of Scotland there was an excellent poultry school and students appreciated very much going there to see what was going on and to hear the whole problem discussed. That facility might be developed quite reasonably by all the schools. There was obviously scope for co-operation with Weybridge and the agricultural institutes and a lot could be achieved if the matter were tackled resolutely.

Mr. W. Tweed said that a packing station was not a slaughterhouse registered or licensed under the Slaughterhouses Act; they were controlled by the Regulations as to Food Hygiene and the Poultry Premises and Vehicles (Disinfection) Order. The inspection of poultry carcases was sometimes done by the management themselves. Often a girl was employed to reject birds which she thought were unfit if the company were anxious not to send out anything sub-standard. This appeared an open field from which a great deal could be learned about poultry diseases.

Recently he had seen a condition in turkeys which had been slaughtered. They had purple discoloration of the skin on the neck, wings and body, and all that could be ascertained was that the birds had recently moulted. Perhaps one of the authors had seen the condition and could comment upon it. It was something that would concern anyone who was carrying out meat inspection.

Mr. Gordon asked whether the rest of the carcase was quite normal and whether the condition was wet.

Mr. Tweed said the condition was seen in the neck and the wings. There were also patches on the carcase which was otherwise normal. There was no subcutaneous oedema or staining but the skin was a purple colour. The birds were confined to coops but it was not bruising. A somewhat similar condition appeared in rabbits and he would like to know if it was commonly met with, was of genetic origin, or only associated with moulting or feather pigment.

Mr. Gordon said that he had seen something similar in chickens.

The Chairman proposed a vote of thanks to the authors and the speakers who had taken part in the discussion, and to all who had worked to make the session a success.

The vote of thanks was accorded with acclamation.

The Profession and the Poultry Industry.—*Concluded.*

teaching schools have not in the past devoted the attention to poultry which this branch of agriculture deserves, but the position is showing some improvement. However, there is little point in teaching a student the cultural characteristics of the *Salmonella* groups or the intricate life-cycle of the *Coccidium* unless he also is given some understanding of the structure of the industry, the highly specialised techniques involved in large-scale chick production, broiler farming and so on which so closely influence the epizootiology of avian disease. It is essential that some attention be given to this aspect of agriculture in the veterinary curricula. Meantime, something can be done by organised refresher courses. We can no longer give other than our best and most highly informed services to this important industry whilst preventing its obtaining help elsewhere. The Veterinary Surgeons Act in protecting us from lay competition imposes the obligation on us to secure the welfare of all animals of whatever species.

Factors Affecting the Solids-not-fat Content of Cows' Milk

BY

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SUMMARY.—Because of the importance of the solids-not-fat (s.n.f.) content of cows' milk, the association between the fat and s.n.f. percentages of milk, and certain factors which can influence the amount of the s.n.f. in milk, are considered. The factors referred to comprise variations due to breed, strain and individuality, and the heritability of the s.n.f. content of milk; age showing a decrease as animals become older; stage of lactation, pointing out a rise due to conception as opposed to a fall in barren cows; the effect of diet, particularly on the protein fraction; the season of the year, with a fall in April and, to a lesser extent, in August; environmental temperature; the influence of hormones; systemic disease; mastitis, causing a reduction, particularly, in the lactose content; and milking technique. Suggested methods of preventing or correcting downward trends are outlined where applicable, and reference is made to the tests used to determine the s.n.f. percentage.

MILK is a mixture of 3 main constituents, water, fat and solids-not-fat (s.n.f.). It is difficult to give an analysis of an "average" sample of cows' milk, but the approximate composition is shown in Table I.

TABLE I

AVERAGE COMPOSITION OF COWS' MILK (PER CENT.)

Water	...	87·6				
Fat	...	3·7				
S.n.f.	...	8·7—	Casein	...	2·6	Protein ... 3·3
			Albumin	...	0·5	
			Globulin	...	0·2	
			Lactose	...	4·7	
			Ash	...	0·7	

The nutritional value of milk is largely derived from the solids which it contains, and it is the s.n.f., and not the fat, content which provides most of the value. The high quality animal protein and the minerals, particularly the calcium, are especially important.

Correlation between the Fat and S.N.F. Content of Milk

There is a tendency for a high fat content to be associated with a high s.n.f. percentage, and *vice versa*, when the milk of one breed is compared with that of another, but this does not always apply to individual animals, and it is not uncommon for cows to produce milk which is above the breed average for one of these constituents and below it for the other. Thus breeding for an increase in fat percentage will not necessarily ensure that the s.n.f. content will also be raised.

There is a correlation between the fat and protein content of milk and Lankamp (1959) estimated by regression coefficients that for a rise in fat of 1 percentage unit, protein rose by 0·31 and s.n.f. by 0·27 percentage units, and for a rise in protein of 1 percentage unit, s.n.f. rose by 0·81 percentage unit. With increasing age the correlation between protein and s.n.f. tended to become less. However, Kleisch *et al.* (1959) estimated the protein and fat content in milk weekly from 17 cows and every 2 days and daily during 2 successive lactations of a pair of monozygotic twins, and they concluded that in individual cases it is not possible to assess the protein content of milk from the fat content.

Breed Differences

Breed variations in the s.n.f. content of cows' milk can be clearly demonstrated, but they are much less than is the case with fat. The breed averages determined by the Milk Marketing Board (1959) for the milk recording year for 1957–8 are given in Table II.

TABLE II

BREED DIFFERENCES IN THE AVERAGE S.N.F. PERCENTAGE OF MILK

Breed	S.n.f. per cent.	Breed	S.n.f. per cent.
Ayrshire ...	8·72	Guernsey ...	8·94
Friesian ...	8·65	Jersey	9·10
Shorthorn ...	8·75	Red Poll ...	8·78

Marked variations are also encountered in strains within breeds, and, in some cases, breeding for volume without regard to quality has resulted in the production of certain families which consistently produce low-quality milk. This has been observed particularly in certain strains of non-pedigree Friesian cattle. By testing individual cows such families can be detected and culled.

Because the breed differences in s.n.f. percentage are not very wide, the introduction of a few cows of the Channel Island breeds into herds of cows of other breeds, yielding low-quality milk, is not a very effective method of raising the herd average.

Individual Variations

Occasionally cows are met which persistently give milk of an unusual composition. This may mean that the milk consistently has a high or low s.n.f. content which cannot be attributed to any of the factors which are known to influence milk composition. Such variations are apparently uncontrollable.

Inheritance

Robertson *et al.* (1956) made a statistical analysis of lactation records from 500 dam-daughter pairs of the Ayrshire breed. Measurements were taken of the yield of milk, fat, s.n.f., crude protein, casein and lactose. The comparatively high heritabilities that they found for s.n.f. and its constituents mean that they should respond directly to selection based on the performance of individuals without progeny testing. But, though fairly certain, the rate of response would be slow because of the low level of culling possible, the low variation between cows in s.n.f. content and the long generation length in the cow. Concentrating entirely on s.n.f. content, a policy of culling the worst third of the first lactation cows and of breeding bulls out of the best 5 per cent. of cows should lead to an improvement of about 0·02 per cent. per year. In the selection of bulls, the high heritability means that a reasonably accurate progeny test for s.n.f. can be obtained with comparatively few daughters, the results for 20 being sufficient.

Genetic selection can only be put into practice if the milk from individual animals can be tested for s.n.f. content. This is particularly important in the case of the dams and grand-dams of bulls standing at artificial insemination centres, which are likely to produce large numbers of daughters. It would also be advantageous for a s.n.f. content requirement to be added to the qualifying standards for a dairy bull licence.

Age

The highest quality milk is nearly always produced by heifers in their first lactation, and the s.n.f. content usually falls slightly but steadily with increasing age. In many animals the decrease in s.n.f. percentage will be about 0·1 between consecutive early lactations and slightly less between consecutive later lactations.

It is advisable to ascertain the s.n.f. content of the milk of first-calf heifers and reject animals giving low results, because it is very unlikely that the quality of their milk will subsequently improve. In herds composed largely of old cows milk with a low s.n.f. content may be produced due to this reduction with age.

Stage of Lactation

Bailey (1952a) surveyed the records from the Shorthorn herd of the National Institute for Research in Dairying for the period 1935 to 1946 and found that there was a drop of about 0·2 per cent. in s.n.f. during the first 2 months of a lactation followed by a period during which there was little change until about the middle of the sixth month. After this there was a difference between pregnant and barren cows, for in pregnant animals there was a steady increase rising by about 0·4 per cent. at the end of the lactation which did not occur in barren animals.

Of the individual constituents of the s.n.f. lactose showed a gradual fall of about 0·5 per cent. from the commencement to the end of a lactation. Once the minimum period was passed the regular fall in lactose was counterbalanced to some extent by increases in protein (Ling, 1956). There is a rapid fall in the chloride content early in a lactation followed by a steady increase which becomes rapid towards the end.

Edwards (1958) found a tendency for substandard milk to be produced by cows which had an interval between calving and successful service of between 195 and 255 days. Between 50 and 56·3 per cent. of samples had a s.n.f. percentage below 8·5 in this group as compared with a range from 29 to 42·9 per cent. for cows which conceived before the 195th day.

If, because of infertility, a number of the cows in a herd are barren and are towards the end of their lactations milk low in s.n.f. may be produced. Such milk may also have a salty taste due to a high chloride content. The only course to adopt is to dry the cows off. If they do become pregnant the milk yielded in the subsequent lactation will again be normal in composition.

Winter Feeding

Rowland (1947) reported a classic experiment carried out at the National Institute for Research in Dairying which showed the effects of 3 diets on the s.n.f. content of milk. Each of the diets was fed to experimental groups of cows for 5-week periods in rotation. The results are given in Table III and it will be observed that the decrease in the s.n.f. per cent. is due very largely to a decline in the protein content. This finding has been confirmed by Comberg and Voigtlander (1958) who studied 149 complete lactations of 88 Black Pied Lowland cows in 16 herds. Some 700 samples of food were analysed, and a statistical study made relating variance of milk components to physiological and environmental changes. It was concluded that food intake influenced the protein and ash, but not the lactose content.

TABLE III

THE EFFECTS OF 3 DIETS ON THE S.N.F. CONTENT OF MILK

Ration	S.n.f. per cent.	Protein per cent.	Lactose per cent.
Normal	8·68	3·07	4·71
Low protein. Full starch equivalent, but only 60 per cent. of calculated protein equivalent needs	8·50	2·95	4·65
Low energy. Full protein equivalent, but only 75 per cent. of the normal starch equivalent ...	8·34	2·86	4·61

The amount and composition of the concentrate ration has been shown to influence the s.n.f. content of milk by other workers. Castle *et al.* (1958) fed Ayrshire cows on a basal ration of 40 lb. of silage and, in addition, 16 lb. of dried grass or hay, each with 2 or 4 lb. of concentrates per gallon of milk produced. There was no effect on the percentage of s.n.f. when dried grass replaced hay, but the higher level of concentrates gave a higher percentage of s.n.f. than the lower. The protein content was significantly lower with 2 lb. of concentrates than with 4 lb. Starodubcev (1959) found that milk protein rose from 3·41 to 3·49 per cent. when the mean protein intake in the diet was raised by 110 g. daily and from

3·46 to 3·67 when it was increased by 547 g. daily. This increase in milk protein was not economical, as it was only obtained by these large increases of protein intake.

The type and quality of the roughage fed during the winter months may have a profound effect on milk composition. Pearce *et al.* (1959) used identical twin heifers in their first lactation to compare the effects of feeding good and poor quality hay. The heifers were allowed 1½ hours restricted grazing, 4 lb. of concentrates per 10 lb. of milk produced, and hay to appetite. The amount of hay eaten, milk production and s.n.f. content of the milk were all significantly lower in the group given poor hay.

It has been suggested that the excessive feeding of silage may adversely affect the s.n.f. content of milk. From mid-January to mid-February Dijkstra (1959) divided Friesian cows into groups of 10, the first group being given daily 44·5 kg. of grass silage and 4·8 kg. of concentrates, the second 22·4 kg. of silage, 6·6 kg. of hay and 5·0 kg. of concentrates and the third 13·2 kg. of hay and 4·8 kg. of concentrates. Group 3 gave 172 g. more s.n.f. and 86·4 g. more protein per cow daily than Group 1, and Group 3 produced more s.n.f. and protein than Group 2. He concluded that silage should not be given as the sole roughage but should be supplemented with, for example, hay. It would appear that the quality of the silage is very important in this connexion. If cows are fed high-quality silage as their only roughage satisfactory results may be obtained, but low-quality bulky silage may lead to the production of milk low in its s.n.f. content.

The quality of dried grass fed can vary milk composition. Campling and Holmes (1958) compared the relative value for milk production of artificially dried cocksfoot and a timothy/meadow-fescue mixture grown both in spring and in autumn. The spring grasses were cut and dried in May and the autumn grasses in September. The rations offered to the individual cows consisted of 17 lb. of one of the grasses under comparison, 28 lb. of fodder beet and 5 lb. of a concentrate mixture for each gallon of milk produced over the first 1½ gallons. The s.n.f. content of the milk was significantly higher on spring-grown cocksfoot than on autumn-grown, and higher, but not significantly so, on spring-grown timothy and meadow fescue than on autumn-grown.

As underfed cows cannot draw on their body reserves for a long period without a decline in milk composition occurring it is important to ensure that cows are adequately fed at all times, and to see that the ration is properly balanced and not deficient, particularly, in starch. Care also needs to be taken to see that they are always fed according to yield, and that the quality of the roughage is not overestimated. Underfeeding during the autumn may result in serious trouble with s.n.f. later in the winter.

Summer Grazing

It is generally considered that good quality grass is a very suitable food for milk production, and that, provided an adequate supply is allowed, milk quality is nearly always satisfactory. In fact the rise in the s.n.f. content of milk which almost invariably occurs when cows are turned out to grass in the spring after being fed on winter rations is probably a good indication of the quality of grass as a food. During dry weather in certain areas the yield of grass may drop so that the cows are underfed, while in the autumn the grass tends to become fibrous and stemmy with a low nutritive value.

It is particularly important that the quantity and quality of autumn grass are not overestimated, and that winter feeding is commenced during October, even though there may appear to be plenty of grass.

Season of the Year

When large numbers of milk samples are analysed throughout the year, so that the effects of factors other than time of year are evened out as far as possible, it is clear that the s.n.f. content of milk varies with the season. There is a drop from January to April followed by a rise in early summer in all districts. Particularly in dry seasons in the drier parts of the country there is another drop during August. Milk Marketing Board (1959) figures showing monthly variations during 1957–8 are given in Table IV.

TABLE IV

MONTHLY VARIATIONS IN THE S.N.F. CONTENT OF COWS' MILK

Month	S.n.f. per cent.	Month	S.n.f. per cent.
October ...	8·81	April	8·67
November ...	8·80	May	8·76
December ...	8·76	June	8·79
January ...	8·72	July	8·72
February ...	8·70	August ...	8·70
March ...	8·69	September ...	8·73

The diet, probably, has some influence on these variations. The drop in late winter may be due to poor quality winter rations, the rise in early summer to the consumption of high-quality grass, and the drop in dry areas in late summer to the decline in grass quality. The steepness of the declines in April and August can be reduced by ensuring that a high nutritional level is maintained throughout the year, and not allowed to drop during these periods. Every effort should be made to prevent the decline starting, as it is usually difficult to check once it has commenced.

Environmental Temperature

A number of workers have shown that at temperatures of 80° F. and over milk yields are reduced, and the s.n.f. percentage is decreased. Such conditions are unlikely to be encountered in the British Isles, and environmental temperatures have little effect in this country.

Hormones

It has been shown experimentally that the administration of oestrogens to lactating cows will produce a marked rise in the s.n.f. fraction of their milk. This has very little practical application as the dosage has

to be carefully controlled and administration can only safely be continued for a limited period. However, it is possible that a short period of feeding a thyroid activating material, such as iodinated casein, might be useful in March when milk quality tends to be low. The spring grass subsequently consumed would tend to check the extent of the decline which would normally follow the cessation of its use.

As oestrogens occur in pasture plants, and are present in the highest concentration during the spring, it is possible that the rise in the s.n.f. content of the milk of cows when turned out on to spring grass may, in part, be due to hormonal activity. Bailey (1952b) has suggested that, as it is known that the production of oestrogens by the pregnant female greatly increases as pregnancy advances, there is, possibly, a correlation between the level of blood oestrogens and the s.n.f. content of milk, and that this may explain the rise in the s.n.f. content of the milk from pregnant animals.

Systemic Disease

Systemic disease may affect the s.n.f. content of cows' milk, and a rise in body temperature is usually accompanied by a decrease in the s.n.f. percentage. However, such conditions largely affect individual cows, and the results have little influence on herd supplies.

Clinical Mastitis

Udder quarters affected with clinical mastitis produce milk which is seriously deficient in s.n.f. In some cases the deficiency is particularly apparent in the first milk drawn from the quarter, and the remaining milk is more normal. However, since only a small percentage of cows are likely to have clinical mastitis at one time, and milk from such quarters is supposed to be, and usually is, excluded from the bulk supply, this type of mastitis does not form an important cause of a low s.n.f. content of milk from a herd.

Sub-clinical Mastitis

This is much more important as milk from affected quarters has a low s.n.f. content but appears normal macroscopically and so is often included in the general milk supply. O'Donovan *et al.* (1960) analysed data from 50 cows mainly infected with *Staphylococcus pyogenes* to determine the effects of udder infections on yield and milk composition. "Infection" meant that pathogenic bacteria were recovered frequently from the fresh milk of the affected quarter, and that milk was positive to the Whiteside test. It was found that lactation yields of milk and s.n.f. were depressed by 10 and 11 per cent. respectively in the presence of infection. These depressions were significant. The mean s.n.f. per cent. of the milk was significantly depressed by $0{\cdot}196 \pm 0{\cdot}092$ during the period of infection. It is suggested that the depressions in lactation yield and changes in milk composition would have been greater in the absence of antibiotic therapy for mastitis. Sub-clinical mastitis affects milk composition by causing a decrease in the lactose and a slight increase in the protein percentages.

Milking

The milking technique and milking efficiency have little influence on the s.n.f. content of milk, but faulty machine milking and errors in milking hygiene can contribute to a high cell count and create a susceptibility to mastitis. The practical importance of this should not be overlooked.

The duration of the intervals between milkings has little effect on s.n.f. If a long night interval results in a lower fat content the percentage of s.n.f. will show a slight increase because, as milk is a mixture of fat, s.n.f. and water, if the percentage of fat is reduced there will be a corresponding increase in the content of the other two. If the percentages are calculated on a fat-free basis this difference disappears.

General

It is very difficult to assess the relative importance of the various factors. Alexander and Leech (1960) made a statistical analysis of the data resulting from a field study of 10 farms in the county of Durham, and showed that age, pregnancy, season of the year and total cell count affected the s.n.f. percentage, and that these effects were additive and independent of each other. From these results no effect associated with nutritional changes could be demonstrated.

The principal effects of the factors, each one freed from effects of other factors, were as follows :—

	S.n.f. *Per cent.*
1. Decline with age from 1st and 2nd to 5 + lactations ...	0·47
2. Mid-lactation fall	0·25
3. Reduction associated with high cell count	0·15
4. Rise from winter to spring ...	0·13

It is generally accepted that high cell counts indicate some degree of udder inflammation. High cell counts occurred both in milk produced by cows that had been cured of mastitis and in milk from cows that had not been affected, and it would appear that mastitogenic organisms do not always cause high cell counts.

Herds in which s.n.f. had been consistently low over a period of years were compared with herds in which s.n.f. had been satisfactory. Analysis of the data showed that about 70 per cent. of the difference in s.n.f. between these groups could be accounted for by differences in age of cow, stage of lactation, cell count and breed.

As a result of a survey of 416 cows in 14 herds, Edwards (1958) found that the lowering of the s.n.f. in the milk was due almost equally to a lowering of the lactose content, in which case it was assumed that mastitis was the main cause, and the crude protein content, due largely to inadequate feeding, and considered, therefore, that mastitis and bad feeding played almost equally important parts in the reduction of the s.n.f. content.

S.n.f. Testing of Individual Cows

One of the great difficulties in determining the cause of a low s.n.f. content in milk in England and Wales

(*Concluded at foot of page overleaf*)

News and Comment

AID FOR THE COLONIAL SERVICE

A Government white paper* of great importance to veterinary surgeons serving in Colonial territories was issued last week and laid before Parliament. Its fundamental provision is to assist territories emerging to self government to maintain an effective professional civil service by providing sufficient funds to make it worthwhile for British civil servants in these territories to remain at their posts. The widespread uncertainty about security of employment, at least for a number of years to come, remuneration and compensation should be allayed in considerable measure. The following is a summary of the white paper's major intentions, but those interested would be wise to obtain the document itself.

The plan will come into effect in most territories on April 1st, 1961, and will remain in operation for about ten years, perhaps more in territories whose need is great. The British Government will financially assist the territories by contributing between £12 million and £16 million each year towards the cost of keeping overseas administrators and technical and professional experts until experienced local staff can be trained to replace them.

* Cmmd 1193. H.M.S.O., price 1s. 3d.

The scheme will cover up to 21,000 pensionable and contract officers overseas. Its advantages will be offered to all existing colonies and dependencies and also to Nigeria (already independent but with many expatriate officers still serving in it), Sierra Leone (due to become independent in April, 1961), and Singapore, and to Northern Rhodesia and Nyasaland. The Bahamas, Bermuda, Brunei and Hong Kong where, for several reasons, this form of help is not regarded as appropriate, will be excluded from the scheme, at least for the time being. Governments which wish to accept the offer will be invited to sign a formal agreement with the United Kingdom Government.

The Paper says, "It is the hope of Her Majesty's Government that these arrangements will substantially ease the difficulties, both political and financial, that confront the governments of developing countries in seeking to employ skilled manpower from overseas in the numbers which their needs indicate but their limited budgets cannot always easily meet."

It declares that as a consequence of the arrangements, even the poorest country should be able to pay the market rate for the expatriate officers they wish to retain. The paper recognises that overseas officers have been worried by uncertainties about

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Factors Affecting the Solids-not-fat Content of Cows' Milk.—*Concluded.*

is that there is not a s.n.f. testing scheme comparable with the existing Milk Marketing Board fat-testing scheme for individual cows. Individual cow tests will often be carried out by dairies on request to help farmers having difficulty in maintaining milk quality, but it is important that facilities for the s.n.f. testing of individual cows in milk recorded herds should be provided as soon as possible. In Scotland the Milk Records Association will test individual cows, and in Northern Ireland the test is carried out regularly on the milk of recorded cows.

Two methods of determining the s.n.f. content of milk are in common use.

1. A gravimetric method in which the samples are evaporated in ovens and the total solids measured by weight. Then the fat percentage, as determined by the Gerber test, is subtracted. This is a reliable test, but expensive equipment is involved, and it is time consuming.

2. The second, widely used method, is to determine the density by a hydrometer and then calculate the s.n.f. from the fat and density. This gives satisfactory readings for bulked milks, but can be misleading if applied to milks from individual cows.

It should be noted that formalin appears to be the only preservative which can be used if milk is to be tested for s.n.f. and it only keeps samples for 2 days. Other preservatives, used to keep samples for longer periods for fat testing, are not suitable for samples to be nalysed for s.n.f.

Considering the relatively small variation in s.n.f. percentage it is particularly important that the analytical results are accurate, so that small differences between animals can be determined. Thus the gravimetric method should be adopted for testing samples from individual animals.

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their future, and that many have tended to leave overseas service as soon as the country in which they served became independent. The new scheme is intended to help them also during this period of uncertainty.

The review of the situation of the Overseas Civil Service which led up to the production of the new scheme also took account of the fact that it has not proved possible to offer alternative employment in government to many officers who have retired from self-governing territories, and of suggestions that one solution would be to create a comprehensive home-based service under which former Overseas Civil Servants would continue to be employed by the United Kingdom Government. Study of these proposals has confirmed, however, that the creation of such a service would not in itself increase the number of jobs available.

An appendix to the paper says: "The creation of a U.K.-based Service as an alternative to the proposals in this paper would convey a false impression of security of employment. The recent proposal by the Select Committee on Estimates for a study of a possible Commonwealth Advisory and Technical Service which is at present under consideration by H. M. Government is for a separate Service on a much more modest scale than the Overseas Civil Service."

The Government considers that through suitable terms of service and an assurance that if officers were retired prematurely satisfactory compensation would be assured, it should be possible to make the overseas service sufficiently attractive to induce most of the officers still needed to remain at work, as well as helping the territories concerned to afford their services. To achieve this end, the Government are prepared to reimburse territorial governments accepting the scheme in this way:

The full cost of inducement allowance designed to attract and keep expatriate officers in service overseas. In this regard, it will be necessary to review the emoluments of officers in many territories;

The full cost of education and children's allowances for the children of officers and of the additional pension or contract gratuity which an officer will earn from his inducement allowance;

Half the cost of passages to and from his home country for the officer and his family. An additional attraction is that this will also include an annual visit to their parents by children being educated overseas;

Half the cost of compensation awarded to an officer for loss of career on premature retirement resulting from the progress to independence of the territory in which he served.

The Government will make no payments direct to overseas officers who, while being members of a common Service under Her Majesty The Queen will continue to owe their full loyalty to the territorial government which employs them.

Dealing with compensation for retiring officers, the white paper details the normal principles to be followed but states that compensation schemes should also provide an inducement to individual officers to continue to serve so long as their services are required. This might be done in a variety of ways such as the payment of compensation by instalments where officers can continue to serve if they wish, or an additional payment to those who elect to stay. The principles to be observed in individual compensation agreements should include the right of every overseas officer to retire with compensation at the time when the United Kingdom Government gives up its responsibility for local Civil Service affairs. The inducement pay of many officers is likely to be increased as a result of the review of salaries.

As to future overseas recruitment, the white paper remarks that the proportion of candidates appointed on contract terms through the Colonial Office has increased markedly in recent years, and the British Government plans wherever possible to obtain recruits on loan from government departments, public corporations and local authorities, so that individuals can be certain of resuming their previous employment upon returning to the United Kingdom.

The paper expresses the hope that service overseas will be "accepted in this country as an asset both to the employer and the employee."

THE ROYAL COLLEGE OF VETERINARY SURGEONS

Temporary Building

The present office of the College at Red Lion Square will be closed at 5 p.m. on the evening of Thursday, December 29th, and will re-open at the new temporary address—College House, Wright's Lane, Kensington, W.8.—on Monday, January 2nd, 1961, at 9.30 a.m.

It is asked that enquiries and correspondence may be kept to the minimum during the week immediately before and after the move.

Wright's Lane runs south from Kensington High Street along the side of Messrs. Pontings. College House is a modern office block on the left towards the bottom of Wright's Lane, when approaching from Kensington High Street. The College accommodation will be on the first floor. Kensington High Street Station (District and Circle lines) is immediately adjacent to the temporary College building.

Library

The packing of the library for removal will commence on Thursday, December 1st, 1960, and the actual removal will be carried out over the week-end commencing Friday, December 16th.

From Thursday, December 1st onwards, until further notice, it will be possible only to provide a limited library service.

As soon as the library in the temporary College building is available to give full services a notice will appear in THE VETERINARY RECORD.

The Old Building

The building and historical contents of 9 and 10 Red Lion Square can be seen by anyone interested, preferably by making a previous appointment, at any time between 9.30 a.m. and 5 p.m. on Monday to Friday inclusive, up to the evening of Wednesday, November 30th.

Special arrangements by previous appointment can also be made for the building to be seen at any time on a Saturday or Sunday until that date.

THE BENJAMIN WARD RICHARDSON LECTURE

On November 25th, 1960, at the Headquarters of the Royal Society of Health, 90 Buckingham Palace Road, London, S.W.1, the Benjamin Ward Richardson Lecture will be delivered by Mr. R. W. Sherwood, Managing Director of the Letchworth Bacon Company. It is entitled "The Equipping and Organisation of Abattoirs," and Professor H. Burrow, Vice-President of the Royal Society of Health, will be in the chair.

In the hope that a number of members of the profession will be attending this meeting it is proposed to hold a meeting of the Association of Veterinary Food Hygienists during the morning of November 25th. All members of the Association, and any members of the profession interested are asked to attend at B.V.A. Headquarters, 7, Mansfield Street, London, W.1, at 10.30 a.m.

AFRICAN HORSE DISEASE

The European Regional Conference has asked the Food and Agriculture Organisation to see if funds can be raised to combat threatened invasions of Europe by African horse disease from the Near East.

The Conference asked the Director-General, Mr. B. R. Sen, to approach European countries and the International Office of Epizootics to investigate the possibility of providing money needed for mass vaccination of horses in the Near East and Turkey. It noted that there was a serious risk of the disease spreading into Europe through the Balkan Peninsula in the near future.

While there is "every indication that there will be a temporary cessation of the infection during the winter months," the Conference report said, the disease will almost certainly appear again in infected countries in the spring of 1961 unless mass vaccination is carried out.

VETERINARY HYGIENE SECTION, R.S.H. CONGRESS

The Royal Society of Health Congress will be held in Blackpool from April 24th to April 28th, 1961. The following programme has been arranged for the Veterinary Hygiene Section, which this year is being held on Wednesday, April 26th, from 2.30 to 5 p.m. under the Presidency of Mr. E. R. Callender, O.B.E., M.R.C.V.S

(a) "Hormones and their effect on Meat and Meat Production" by S. Jennings, M.R.C.V.S., University of Glasgow Veterinary School, President of the British Veterinary Association.

(b) "The Changing Emphasis in Meat Inspection" by R. V. Blamire, M.R.C.V.S., D.V.S.M., Deputy Chief Technical Adviser, Ministry of Agriculture.

The Secretary of the Section is Dr. J. C. Greatorex of the Royal Veterinary College Field Station, North Mimms, Hatfield, Herts.

B.V.A. CONFERENCE ON HYPOMAGNESAEMIA

As previously announced in THE VETERINARY RECORD this two-day Conference will be held at the Victoria Halls, Southampton Row, London, W.C.1, on November 23rd and 24th. Papers which are to be circulated in advance have now been received and are in process of printing. Those who are organising the Conference and who have, therefore, read the papers find them of the highest excellence and of great interest and importance to the practising veterinary surgeon as well as to others interested in the broad field of animal nutrition and management. A great deal of work is being put into this Conference, and it is hoped that members will support it in the largest possible numbers, so as to ensure its success and encourage the B.V.A. to envisage other Conferences along the same lines. Space this week does not allow us to set the programme out in the customary full form, but its details are as follows:—

Opening remarks by the Conference Chairman, Mr. Sydney Jennings, President of the B.V.A.; followed by a General Introduction by Professor A. Robertson; next 3 papers: *Basic Science of Magnesium Metabolism* by Dr. Thomas Douglas, *The Absorption and Excretion of Magnesium in the Ruminant* by A. C. Field; and *Intake and Utilisation of Magnesium from Herbage by Lactating Cows* by A. Kemp.

After a discussion and break for luncheon J. B. White will read his *General Paper on Hypomagnesaemic Tetany*, followed by short papers from the following members in general practice; J. C. MacKeller, A. Nisbet, A. Steele-Bodger, K. P. Anderson, W. J. B. Robson and J. P. Pickering. The remaining papers will be *Factors involved in Hypomagnesaemia in Sheep* by Professor J. S. S. Inglis; *Factors Influencing the Incidence of Hypomagnesaemia* by A. Thomson and Professor M. L. 't Hart; and *Prevention of Disease* by Dr. Ruth Allcroft. Mr. S. L. Hignett, Senior Vice-President of the B.V.A., will sum up.

Mr. G. H. LOCKE

Mr. G. H. Locke, whose death was announced with deep regret last week, was born in Manchester on December 21st, 1875, the eldest son of Sam Locke, M.R.C.V.S. He was educated at Claremont College, Blackpool, and the New Veterinary College, Edinburgh, from which he graduated on December 21st, 1896, his 21st birthday. He immediately joined his father in general practice in Manchester, and on the death of his father in 1910, during the latter's term of office as President R.C.V.S., he continued the practice until his retirement in 1951.

Mr. Locke joined the Lancashire Division of the N.V.M.A., and also the parent body as soon as possible after graduation and continued to be a keen supporter of both bodies all his life. He was elected hon. secretary of the Lancashire Division in 1899, relinquishing that position in 1914 when he was elected President, continuing in office until the end of the 1914-18 war.

For many years an active member of the Council of the N.V.M.A. he acted as local secretary when

the Annual Congress was held in Manchester. He was later to be elected Vice-President of the Association, and, a few years ago, became an Honorary Life Member.

He was elected to the Council of the R.C.V.S. in 1920 and served continuously from that date until 1940. In 1934-5 he became President of the College, and during an eventful year of office, was presented with the Royal Silver Jubilee Medal, and also led the British delegation attending the International Veterinary Conference in New York. Another of his interests was the National Veterinary Benevolent and Mutual Defence Society which he served for very many years in the dual offices of hon. secretary and hon. treasurer.

During the South African War Mr. Locke travelled extensively in Austria and Hungary examining horses and mules for the Remount Commission; during the Great War of 1914-18 he was similarly engaged in Canada and the United States.

He was for many years a Local Veterinary Inspector of the Ministry of Agriculture & Fisheries, and held similar appointments to the Lancashire County Council and other local councils; he was resident veterinary surgeon to the Royal Lancashire Show Society, and also, on many occasions, to the Shire Horse Show. He acted as veterinary surgeon to the Belle Vue Zoological Gardens, Manchester.

His interest in Freemasonry was lifelong and he attained Provincial honours. He even attended a meeting as a guest of Mr. J. M. Ingram the week before his death—the occasion coinciding with his 50th anniversary as a Mason.

All his life he was a lover of sport, particularly cricket and association football. In this connexion he had the remarkable record of maintaining continuous membership for over 50 years of the Lancashire County Cricket Club and the Manchester City A.F.C. Both clubs elected him to Life Membership.

In January, 1900, he married, and with Mrs. Locke celebrated their Diamond Wedding last January. He left three sons and one daughter, eight grandchildren and one great-grandchild.

HISTORY OF THE R.A.V.C.

The following further donations have been received: Major R. I. Macrae, M.R.C.V.S., £1; The Wellcome Trust, £500; Major T. Boundy, B.V.SC., £2 2s.

DRUG RESEARCH IN 1959

The latest survey by the Association of British Pharmaceutical Industry shows that over £6,257,025 was spent on research into and the development of new and improved drugs by members of the association last year. This is an increase of more than £1m. on 1958 and double the amount spent in 1954.

PERSONAL

Births

FITCHIE.—On October 25th, 1960, to Lynne, wife of Peter Fitchie, B.V.M.S., M.R.C.V.S., Llanfyllin, Montgomeryshire, a daughter, Carolyn Eve.

WATKINSON.—On September 29th, 1960, to Jessie, wife of Jack Watkinson, B.V.M.S., M.R.C.V.S., 8, St. Mary's Mount, Leyburn, Yorks., a son, John Lindsay, brother for Helen and Jane.

CROOKS.—On October 29th, 1960, to Heather, wife of J. L. Crooks, M.R.C.V.S., a daughter, Elizabeth, sister for Annette and James.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

JENKINS, Francis Henry, of 2 Tor-o-Moor Road, Woodhall Spa, Lincs. Graduated London, December 16th, 1890. Died October 12th, 1960.

COMING EVENTS

November

9th (Wed.). EASTERN COUNTIES VETERINARY SOCIETY. Autumn Meeting at the School of Veterinary Medicine, Madingley Road, Cambridge, 11 a.m.

B.S.A.V.A. METROPOLITAN REGION. Meeting at the Royal Society of Medicine, 1 Wimpole Street, London, W.1, 7 p.m.

DUMFRIES AND GALLOWAY DIVISION. Autumn Meeting at the Queensberry Arms Hotel, Annan 2.15 p.m.

SOUTHERN COUNTIES VETERINARY SOCIETY. Meeting at the Red Lion Hotel, Salisbury, 7.30 p.m.

10th (Thurs.). B.S.A.V.A. SUSSEX DIVISION. Meeting at the Ciba Laboratories, Horsham, 8 p.m.

11th (Fri.). V.V.B.F. NORTH OF SCOTLAND DIVISION. Annual Dinner-Dance in the Northern Hotel, Aberdeen, 7.30 p.m.

ROYAL VETERINARY COLLEGE ASSOCIATION. A.G.M. at Royal Veterinary College, Camden Town, N.W.1, 6 p.m., followed by the 23rd Commemoration Ball at the College, 8 p.m. to 2 a.m.

17th (Thurs.). DUMFRIES AND GALLOWAY V.V.B.F. LADIES GUILD. Annual Buffet Dance at the Cairndale Hotel, Dumfries, 8 p.m.

V.V.B.F. COUNTY OF AYR LADIES' GUILD. Annual Buffet Dance in Western House, Ayr.

23rd (Wed.). WESTERN COUNTIES VETERINARY ASSOCIATION. Annual Dinner Dance at the Imperial Hotel, Exeter, 7 p.m.

23rd and 24th (Wed. and Thurs.). B.V.A. CONFERENCE ON HYPOMAGNESAEMIA. Meetings at the Victoria Halls, Southampton Row, London, W.C.1, commencing 10.30 a.m. each day.

24th (Thurs.). ROYAL (DICK) VETERINARY COLLEGE RUGBY CLUB. Annual Ball in the Adam Rooms, George Street, Edinburgh, 9 p.m.

30th (Wed.). A.V.T. & R.W. SCOTTISH REGIONAL GROUP. Meeting at the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

December

2nd (Fri.). NORTH OF ENGLAND VETERINARY MEDICAL ASSOCIATION. Annual Dinner-Dance at the White Swan Hotel, Alnwick.

B.V.A. COMMITTEE AND COUNCIL MEETINGS

Tuesday, November 15th, at 7, Mansfield Street
5 p.m. Finance Sub-Committee.

Wednesday, November 16th, at 7, Mansfield Street
10.30 a.m. Small Animals Committee.
12 noon Parliamentary and Public Relations Committee.
2.15 p.m. Veterinary State Medicine Committee.
4 p.m. Home Appointments Committee.

Thursday, November 17th, at 7, Mansfield Street
10.30 a.m. Farm Livestock Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, November 18th
10.15 a.m. Council Meeting in the Connaught Rooms, Great Queen Street, London, W.C.2.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ches. Big Bailey Riddings Farm, Penningtons Lane, Gawsworth, Macclesfield (Oct. 28).
Notts. Lowes Farm, Yard, Stoke Bardolph, Nottingham (Oct. 28).
Salop. New House Farm, Wollerton, Market Drayton (Oct. 27).
Somerset. Mollans, Edgarley, Glastonbury (Oct. 31).
Wilts. Twatley Manor Farm, Malmesbury (Oct. 31).

Fowl Pest

Cambs. 31 Westfield Road, March (Oct. 25); 9 Glebe Close, Manea, March (Oct. 30).
Essex. Ramsworth, Perry Lane, Langham, Colchester (Oct. 26); Ivy Lodge Farm, Harold Wood, Romford (Oct. 27).
Lincs. 11 Russell Avenue, North Hykeham (Oct. 26); Home Farm, Thorpe-on-the-Hill (Oct. 30).
Mons. Ty Isif Farm, Llantarnum, Cwmbran (Oct. 27).
Norfolk. Dysons Farm, Tibenham, Norwich; Whitehouse Farm, Lone Row, Tibenham, Norwich; Heath Farm, Banham, Norwich (Oct. 25); Greenhouse Farm, Winfarthing, Diss; Red Lodge, Dodds Road, Attleborough; Long Row Farm, Tibenham, Norwich (Oct. 26); Hillside, Hingham Road, Great Ellingham, Attleborough; Finchams Farm, North Lopham, Diss (October 27); Low Street Farm, Wicklewood, Wymondham; Preston Green Farm, Tibenham, Norwich; Crown Farm, Banham, Norwich (Oct. 28); The Hollies, Morningthorpe; 204 Spixworth Road, Old Catton, Norwich; White House Farm, Banham, Norwich; Lilac Cottage, Low Road, Tibenham, Norwich; Honeysuckle Cottage, The Green Wilby; Quidenham, Norwich (Oct. 29); Fox Farm, Carlton Rode, Norwich; Walnut Tree Farm, Winfarthing, Diss (Oct. 30); Church Farm House, Banham; Heath Cottage, Heath Road, Banham (Oct. 31).
Salop. Bodey House, Stanton, Shrewsbury; 5 Wytheford Road, Shawbury, Shrewsbury (Oct. 29).
Somerset. Victoria Farm, Bradley Lane, Glastonbury (Oct. 25); The Post Office, West Pennard, Glastonbury; New Road Farm, West Pennard, Glastonbury (Oct. 26); Netherha, Over Stratton, South Petherton, Stratton House, Over Stratton; South Petherton; Greystones, Coat, Martock (Oct. 27); Mead Farm, Seavington, Ilminster, Station Hill Farm, Misterton, Crewkerne; 59 Lower Street, Merriott, Crewkerne (Oct. 31).
Suffolk. Northcote Avenue, Bury St. Edmunds (Oct. 26).

Swine Fever

Beds. Park Farm, Bletsoe, Bedford (Oct. 27).
Ches. Bowdon View Farm, Yarwood Heath, Altrincham (Oct. 31).
Denbigh. Ty'n-y-groesffordd Farm, Llanbedr, Ruthin (Oct. 25).
Derbys. Algreave Hall Farm, Shipley, Ilkeston (Oct. 26).
Durham. Brookside Farm, Bishopton, Stockton-on-Tees; Ketton Hall, Coatham, Mundevill, Darlington; 5 Hargreave Terrace, Darlington (Oct. 26); Allotment House, Witton-le-Wear, Bishop Auckland (Oct. 27); Littleburn Farm, Langley Moor (Oct. 28).
Glams. Piggeries, Brynglas Farm, Ynysmeudwy, Pontardawo, Swansea (Oct. 31).
Kent. Scriventons, Speldhurst, Tunbridge Wells (Oct. 28).
Lancs. Lane End Farm, Mawdesley, Ormskirk; Hill Top Farm, Jeppes Lane, Barton, Preston (Oct. 27); Westby Hall Farm, Westby with Plumpton, Kirkham, Preston (Oct. 28).
Lincs. Manor Farm, Rowston (Oct. 26); Campbells Farm, Gunness, Scunthorpe (Oct. 28).
Norfolk. Hall Farm, Lords Lane, Burgh, Castle, Gt. Yarmouth (Oct. 31).
Notts Whitehaven Farm, Mansfield Lane, Calverton (Oct. 31).
Staffs. Deanery Farm, Church Road, Shareshill, Wolverhampton (Oct. 31).
Suffolk. Oak Glen, Bramfield, Halesworth (Oct. 28).
Wilts. Mere Down Piggeries, Mere, Warminster (Oct. 25).
Worcs. Churchill Farm, Beoley, Redditch (Oct. 28).
Yorks. Old Trent Road, Beckingham, Doncaster; Woodmans Cottage, Fenwick, Askern, Doncaster; Blackhorse Hotel, Swainby, Northallerton; Beck Hill Farm, Scorton, Richmond (Oct. 27); Cote Farm, Oxenhope, Keighley; Bainesse Farm, Catterick, Richmond; Bridge Farm, Catterick, Richmond (Oct. 28); High Grange Farm, Exelby, Bedale (Oct. 31).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SCIENTIFIC

Resistance to *Trichostrongylus colubriformis* Produced by X-irradiated Larvae

Sir,—I refer to the letter from Dr. W. F. H. Jarrett and his co-workers in your issue of October 15th. The results recorded in their letter appear open to another, simpler explanation than the one they offer. This is that exposure to X-rays killed some of the *T. colubriformis* larvae with the result that the "vaccinated" animals were receiving a dose of some viable larvae, and some larvae which were rendered non-viable by the X-rays, and played no further part in the events recorded. It is as reasonable to suppose that experience of the infestations resulting from 2 such doses of, say, 2,000 fully viable larvae would confer some degree of immunity as to postulate that this immunity resulted from the larvae being damaged by the X-rays.

It can still be argued that these "viable" larvae gave rise to imperfect adult females—females that were fully immunogenic but of reduced reproductive capacity. Experience with other species suggests that one month from ingestion of larvae to slaughter is a very short period on which to base a judgment

of the behaviour of a population. The great range of response to a given challenge in a seemingly homogeneous group suggests that great caution is necessary in comparing arithmetic mean values of worm populations of small groups. But even if these reservations are set on one side, several questions on the data presented on egg counts require answers. These are:—

What were the respective ages of the lambs used as controls on the infectivity of the larvae (para. 3) and the 15 plus 5 lambs considered in para. 4? It is not possible to ascertain from the text whether these 2 groups were of the same age and so could be expected to show similar egg outputs. Comparison is difficult in any case since in para. 5 the egg count data appear to be drawn from 10 animals and the worm burden data from 5 only.

What treatment did the 5 control lambs of para. 4 receive and what was their egg output? It is not clear whether these lambs had had previous experience of worms. Controls on the immunogenic capacity of X-irradiated larvae should have received, on the basis of the ideas of Dr. Jarrett *et al.*, 2 doses of 10,000 normal larvae 6 and 7 months previously, or, on the basis of the interpretation I am offering, 2 doses of, say, 3,000 normal larvae. On the other hand, if they had no previous experience of infection, their egg count data would have furnished evidence on the levels of output to be expected from undamaged female worms.

How was the " mean post-vaccination egg output " of para. 5 determined? It does not seem possible to compare this *mean* with the other *peaks* (paras. 3 and 4). Here again the egg output appears to have been determined from 10 lambs and the worm burden from an unstated fraction of 10. Presumably 5 were slaughtered to determine the worm burden developing from the vaccine, and 5 to determine the worm burden resulting from the challenge dose.

I cannot see any reliable support in the data presented for the statement that worms developing to the adult stage from X-irradiated larvae were largely " non-egg laying females."

Yours faithfully,
M. G. CHRISTIE.

Parasitology Department,
Animal Diseases Research Association,
Moredun Institute,
Gilmerton, Edinburgh, 9.
October 29th, 1960.

GENERAL

Veterinary Education

Sir,—Professor Weipers' stimulating Congress paper about veterinary education is indeed timely. The problems which are posed by the overcrowding of curricula and the rapid development in the scope of the profession are not such as may be solved by the addition of a year to the degree course or by insisting on a sufficiently high standard of University entrance that the 5-year course could begin in the 2nd year as it were.

Such expedients can at best give a very short breathing space in which to introduce new ideas. The 4-year course lasted for half a century or so; the 5-year one, more or less 25 years; a 6-year course could give us barely 10 years in which to solve our problems and I think everyone would agree that a 7-year basic degree course or its equivalent is unthinkable. Professor Weipers' suggestions to increase efficiency by integrated teaching aimed at producing a much better adapted type of professional scientist, and the provision of post-graduate specialist qualifications are, in themselves, excellent but one wonders if they go anything like far enough.

The methods of teaching used in the universities differ little from those used in the kindergarten and it seems to me that throughout the period of education we are far more preoccupied with turning out graduates loaded with a burden of facts than with ensuring that primarily they have learned how to think for themselves.

Surely our aim should be not to produce a professional man who conforms more or less to a set pattern, but to achieve a certain attitude of mind in our University graduates?

If a boy has been taught how to learn at school so that he can easily acquire and sift for himself the information with which the standard text-books are crammed, and if he has also developed an inquisitive turn of mind, his teachers at the university can concentrate upon the fundamentals of his discipline in a largely tutorial capacity instead of spoon-feeding him with countless dribbles of incidental detail to be mugged up for an examination.

A system of teaching based upon work in the laboratory and clinic rather than in the lecture theatre could give the student a far stronger grasp of the fundamentals of his subject provided that he approached it in the right frame of mind. In the course of his studies he would, incidentally, acquire a large amount of detailed information which, of course, he would have to augment by his own efforts. At the end of the course the student would have a graduate a little less full of facts perhaps (many of them to be forgotten anyway) but with a properly trained mind; whereas the spoon-fed graduate must start to become out-dated on the day of his graduation with little chance to adapt himself to changing conditions in the 40-odd years of work which lie ahead unless, of himself, he has acquired the necessary qualities of mind.

Nearly all our work, whether as diagnosticians or as research workers, consists of the learning process, that of asking questions and answering them satisfactorily from our own or other people's observations. Yet, in the degree courses as set out to-day, the questions are almost exclusively asked and answered by the teachers rather than the students. Professor Weipers has stressed with great force the truth of the saying that more mistakes are made by not looking than by not knowing: yet our graduates leave the universities with far more knowledge of the data to be considered when arriving at a particular diagnosis than of the fundamental methods by which these data must be obtained. Whilst a few people possess almost uncanny powers of observation, most of us need to develop our natural powers in this direction enormously before

we can become really able diagnosticians; for who would deny that the ability to notice what one sees is at the root of good work in the laboratory, the clinic and the field? Yet in how many final examinations does the power of personal observation or lack of it significantly affect the assessment of a student's capabilities?

The small compass of a letter is no place to discuss new proposals for teaching in any detail, but the kind of system I have in mind would be similar in principle to that employed at Liverpool for teaching practical anatomy, necessarily adapted to other disciplines and carried to its logical conclusion. For those unfamiliar with the system it is briefly this: the practical work of the course is divided into a large number of small serial sections, and having completed each section the student must satisfy his teacher that he is properly familiar with it before proceeding to the next.

This tutorial approach has the disadvantage of requiring a larger staff, but properly carried out it forces the student to work consistently instead of cramming hard for the last months before an examination. Additionally, by giving the teacher a very close insight of the day-to-day progress of each student it lessens the importance of professional examinations in assessing a student's abilities. Thus, perhaps half of the total marks could be awarded on class work and the other half on a formal examination. The external examiner would remain as an essential watchdog and co-ordinator of teaching standards between different colleges, and the dreaded examination would be cut down to something like its proper size. I think we would all agree that the principal thing which an examination assesses is a candidate's ability to pass examinations.

To my mind the solution to our difficulties lies in a re-thinking of our methods of education at least from G.C.E. ordinary level onwards, and preferably from much earlier; for whilst our teaching methods consist primarily of driving facts into a pupil's head like nails into a door, the process of learning must necessarily remain a very inefficient one. I, for one, would be the last to deny that post-graduate training in specialist subjects must come in the near future, but this in no way lessens the overriding need to provide professional men equipped not with empiricism but with trained minds.

Yours faithfully,
A. S. WALLIS.

Ministry of Agriculture, Fisheries and Food,
New Haw,
Weybridge,
Surrey.
October 26th, 1960.

Sir,—Having read the letters from Mr. Pickering and Miss Joshua, I find myself, like Mr. Spratling, a little perturbed that critical opinions on the quality of teaching at veterinary schools should be expressed without first-hand experience of that teaching. I am quite ready to concede that there are good and bad teachers just as there are good and indifferent practitioners; but I cannot see the logic in the suggestion that students should have inflicted upon them even more lectures, no matter of what quality, than they are required to suffer at present. A reduction in formal teaching would be a more salutary innovation both from the students' and the teachers' points of view.

If anyone believes that routine teaching is attended by an atmosphere of glamour whereby the lecturer is afforded a platform and guaranteed an audience while he displays his histrionic ability let me hasten to disillusion him or her. As Mr. Spratling rightly points out, the steady pursuit of the annual programme of routine instruction both in the lecture room and the practical classroom provides a greater challenge than the graduate would meet in private practice.

If practitioners truly have the interests of veterinary education at heart, they can best serve this purpose by ensuring a steady flow to the clinical teaching departments of more suitable teaching material than the difficult and unrewarding cases which have in the past been jettisoned upon us. Give us the type of case the graduate is likely to meet as a rule in practice and we can be sure of training the undergraduate to meet his problems in a reasonable and satisfactory manner.

Yours faithfully,
E. F. LEWIS.

21, Byng Drive,
Potters Bar,
Middlesex.
October 31st, 1960.

Survival Despite an Imperforate Anus

Sir,—In a recent issue (September 3rd) Mr. Bryan Boswood reported survival for a fair time of cases of atresia ani in piglets. Such cases have been seen by me on several occasions at the age of 4 weeks and older. Some colleagues in this country have encountered piglets surviving even longer. These cases appear to thrive only slightly less well than their litter mates, the most marked difference being enlargement of the abdomen. It appears, therefore, that the consensus on the matter of survival of piglets with atresia ani is too conservative.

Yours faithfully,
C. F. B. HOFMEYR,
Professor and Head, Dept. of Surgery.

Faculty of Veterinary Science (University of Pretoria), Onderstepoort Veterinary Research Laboratory,
P.O. Onderstepoort,
South Africa.
October 18th, 1960.

ADVERTISER'S ANNOUNCEMENT

SMITH KLINE & FRENCH LABORATORIES LTD., announce that "Bifuran" Soluble Tablets for the treatment of coccidiosis in poultry and enteritis in pigs are now available in new containers and in a new pack size. Future supplies will be packed in cylindrical aluminum containers of 50 and 250 tablets. These replace the bottles of 25 and 250.